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FACTORS IN THE REDUCTION OF MORTALITY FROM PULMONARY ABSCESS*

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SINCE the time of Hippocrates[1] reports concerning the management of lung abscess have appeared periodically in the medical literature. In the last two decades the frequency of these reports has increased greatly, but their substance, so far as results are concerned, remains much the same — to the distress of all who read of failure to treat adequately this serious disease. There has been a failure in spite of the tremendous amount of research that has been done on the subject, in spite of vast improvements in diagnostic facilities and in spite of great progress in the development of therapeutic measures, including the recently introduced chemotherapeutic agents.

In making a critical study of the extensive literature that has accumulated, one cannot help being struck by several salient facts. The first is the failure of conservative treatment alone in all well-controlled series. This is meant to include the application of the following measures, either alone or in various combinations: supportive therapy, drug therapy, postural drainage, bronchoscopic aspiration and pneumothorax treatment. The dismal results obtained by these measures are presented in Table 1. The second is the uniform, almost stereotyped conception of leading authorities about the proper management of the patient with abscess of the lung. This conception is, essentially, that such a patient should be treated by a strictly conservative and supportive regime for at least six to sixteen weeks, or until he proves that he needs additional aid by his rapidly declining course, which threatens to terminate fatally under the existing form of treatment. Only after this trial period, or after the patient has run his swiftly failing course, should surgery be carried out. This plan of treatment has not been significantly altered in the last forty years. In practice this combined management has also failed, as Table 2 shows. Such high mortality rates and low cure rates condemn this type of management, which has been given adequate trial. The third striking fact encountered is that unquestionably surgery in its broadest meaning must also be classed with

TABLE 1. *Results of Medical and Miscellaneous Treatment by Methods Other Than External Drainage of Abscess.*

YEAR	AUTHORITY	No. OF CASES	RESULTS: DEAD	RESULTS: LIVING WITH DISEASE, IMPROVED OR UNIMPROVED	RESULTS: CURED
			%	%	%
1898	Withington[2]	36	56	33	11
1919	Lord[3]*	97	74	18	7
1932	Smith[4] (collected cases)	940	40	—	—
1934	King and Lord[5]†	114	35	25	40
1935	Kline and Berger[6]				
	Abscess	12	58	—	—
	Gangrene‡	41	44	44	13
1940	Rosenblatt[7]§	73	47	47	7

*Theoretical estimate based on 97 cases.
†Calculated from published results in medically treated group; no correction made for inevitably fatal cases that were transferred to surgeons.
‡Calculations based on published chart, excluding cases classed by authors as bronchitis and pneumonitis.
§Calculated from author's tables, surgically treated cases excluded.

TABLE 2. *Results of Generally Accepted Plan of Combined Medical and Surgical Treatment.*

YEAR	AUTHORITY	No. OF CASES	RESULTS: DEAD	RESULTS: LIVING WITH DISEASE, IMPROVED OR UNIMPROVED	RESULTS: CURED
			%	%	%
1929	Flick, Clerf, et al.[8]	161	14	36	50
1934	King and Lord[5]*	210	37	28	35
1934	Brunn[9]†	205	50	50	
1936	Allen and Blackman[10]‡	94	30	17	53
1936	Cutler and Gross[11]	85	38	20	42
1938	Rives, Major and Romano[12]	239	42	—	—
1940	Sweet[13]	124	34	17	49

*Calculated from authors' figures.
†Cases classified by author only as "improved" or "dead." Undoubtedly cures were included in "improved" group.
‡Cases in which abscess was secondary to bronchiogenic carcinoma are omitted.

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the therapeutic measures that have failed. In a group of 2114 collected cases of surgically treated lung abscess, reported by Allen and Blackman,[10] the mortality rate was 34.2 per cent. We believe that future statistics will prove that surgery will continue to fail so long as this term is used to include a multitude of operative procedures, the value of which has been conclusively disproved by uniformly bad results.

Inasmuch as practically all the reports in the literature were based on hospitalized cases, it caused us to wonder whether some patients recover from abscess of the lung without being admitted to an institution, and whether the disease is actually so serious as the reports led us to believe. To determine whether or not this was true, we made a survey of 100 patients with lung abscess, proved by clinical history and chest roentgenograms. This study was made possible by the co-operation of various diagnostic clinics, sanatoriums and private offices, where patients in all stages of the disease are seen, including some patients who recovered without hospitalization. However, much to our dismay, we found that even in this highly representative group of cases the results were not far different from those in the other reported cases. Thirty-one patients died of the disease. Persistent symptoms were present in 20 of the group. Forty-nine were asymptomatic.*

What, then, is the answer to this problem? To date the most hopeful solution is found in the reports coming from Dr. Harold Neuhof and his associates, in which lung abscess is treated as a surgical problem from the onset, and immediate and early drainage is employed. The most recent publication from his clinic[14] concerns the results of this type of treatment in 104 consecutive cases. There were 4 deaths in the series, and the vast majority of the survivors were cured. It is our firm belief that anyone can obtain similar results if, and only if, he abides by the sound pathological and surgical principles on which this excellent work is founded.

The purpose of this paper is to present our conception of the proper management of lung abscess, based largely on the results obtained in treating 95 cases in the last eight years. In the group of patients treated by various generally accepted therapeutic measures, excluding external drainage and excision, the mortality rate was 53 per cent, and the cure rate 26 per cent. In the group of patients treated by lung resection the mortality rate was 23 per cent, the cure rate 62 per cent. In the group treated by external drainage at an advanced stage of the disease the mortality rate was 32 per cent, and the cure rate 26 per cent. *In the group of patients treated by external drainage at an early stage of the disease the mortality rate was 6 per cent, and the cure rate was 94 per cent.* The cases in the latter group are comparable to the ones reported by Neuhof and Touroff.[14]

*Cure was not used because final roentgenograms to rule out the presence of persistent infiltration were not available in many cases.

Definition

Lung abscess may be defined as any suppurative infection with cavitation, arising primarily as a result of implantation of a group of pyogenic organisms in the periphery of the tracheobronchial tree. This therefore includes all such conditions described in the literature as "pulmonary abscess" or "pulmonary gangrene," many cases described as suppurative disease of the lung" or "pulmonary suppuration" and cases of "acute putrid abscess of the lung" or "aputrid abscess." We have excluded only the following types: those primarily a part of a general pyemia; those which are tuberculous; those secondary to unquestioned primary bronchiectasis; and those secondary to pulmonary tumors.

Practical Pathological Considerations

Several fundamental pathological considerations are of definite practical value and therefore deserve discussion.

Bacteriology

Our bacteriologic studies have been of help in a single but important way. They have emphasized the generally accepted fact that the majority of lung abscesses are caused either by strictly anaerobic organisms or by a combination of aerobes and anaerobes. It is therefore imperative to establish early and adequate oxygenation of the abscess cavity, if the concentric invasion of the histolytic bacteria is to be stopped. The consistent and dramatic conversion of a ragged, putrid-smelling abscess cavity into a clean, odorless, granulating wound in a short period, after the use of zinc peroxide paste, has demonstrated to us the value of adequate oxygenation. The rapid reversal of this reaction during the early postoperative period, when the chemical is omitted, indicates that it is very beneficial to supply even a greater amount of oxygen than an adequate external drainage tract affords.

Location

The location of an abscess, with relation to the surface of a pulmonary lobe in which it is situated and with relation to the limiting surfaces of the pleural cavity, is of great importance. The position of 91 abscesses in our group was definitely

determined (Table 3 and Fig. 1). All the abscesses were located peripherally with regard to the lobe in which they were situated. Furthermore, a portion of the wall of the cavities was in juxtaposition to the surface of the pulmonary segment. Thus in the vast majority of cases the abscesses were readily accessible for surgical drainage.

TABLE 3. *Location of 91 Abscesses.*

LOCATION	RIGHT LOBES				LEFT LOBES			ALL LOBES
	UPPER	MIDDLE	LOWER	TOTAL	UPPER	LOWER	TOTAL	
Posterior	6	0	23	29	7	24	31	60
Lateral	6	0	0	6	6	0	6	12
Anterior	2	2	0	4	1	0	1	5
Mediastinal	1	0	0	1	0	0	0	1
Paravertebral	0	0	3	3	0	1	1	4
Interlobar	2	1	1	4	4	1	5	9
Totals	17	3	27	47	18	26	44	91

Condition of Overlying Pleura

The presence or absence of pleural symphysis, overlying the abscess, has a vital bearing on surgical management. We were able to determine definitely the condition of the pleura overlying 84 abscesses. With only three exceptions, pleural symphysis between lung and chest wall was present (Table 4). In two of the three exceptions the abscesses pointed on the interlobar fissure, where adhesions were present. It is noteworthy that adhesions were present just as consistently in the group of lesions under six weeks in duration as they were in the group of greater duration. Since all abscesses are almost invariably located peripherally and since pleural symphysis develops at an early stage of the disease, it is possible to employ external drainage early and as a single-stage procedure, with the provision that localization is accurate.

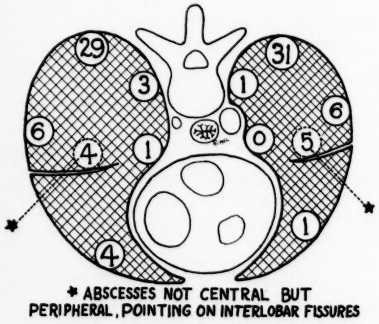


FIGURE 1. *Diagram Illustrating Location of 91 Abscesses in Respect to Lung Boundaries.*

Each number indicates the frequency with which lesions were found in each location. Sixty of the lesions were in the vertebral gutter. Forty-seven of these, or 51 per cent of the entire number, were in the apex of the lower lobe. (See Table 3 for lobe distribution.)

Contents of Cavity

To us the most outstanding pathological consideration concerns the contents of the cavity. In the great majority of cases of abscess of the lung the cavity contains not only fluid pus, but also a semisolid slough of necrotic lung tissue and debris. The presence of solid debris within the cavity has been mentioned by previous writers, but the role it plays in the prevention of solid healing of the cavity has not been emphasized. It is one of the principal causes of failure of all therapeutic methods based on internal drainage. This mass of amorphous debris interferes with oxygenation of the cavity and obstructs drainage of the communicating bronchus. In a small percentage of cases the bronchial opening is large enough, or the slough becomes sufficiently liquefied, to permit its evacuation via the bronchus. Should this mass find an exit, complete recovery with conservative methods of treatment may follow. However, in the vast majority of cases the bacteria-laden mass becomes more or less inspissated and encapsulated, acting as a foreign body that prevents union of cavity

TABLE 4. *Condition of Pleura over 98 Abscesses.*

CONDITION OF PLEURA	CASES UNDER 6 WEEKS' DURATION	ALL CASES
Abscesses pointing on periphery of lung:		
Pleural symphysis present	24	75
Pleural symphysis absent*	1	1
Undetermined	5	13
Totals	30	89
Abscesses pointing on interlobar fissures:		
Symphysis between lobes, no symphysis to chest wall		1
Interlobar empyema, no symphysis to chest wall		1
Interlobar empyema, symphysis to chest wall		6
Undetermined		1
Total		9

*Abscess secondary to aspiration of foreign body.

walls, and solid healing fails to take place. Not only does it prevent solid healing, but it acts as a bomb, teaming with virulent and invasive bacteria, which constantly menaces the life of the patient until it is removed. At any time the delicate balance between the patient's resistance and the destructive force of the bacteria may be upset, and the patient may develop one of the fulminating exacerbations so characteristic of lung abscess. A patient, even though asymptomatic, should never be considered cured and out of danger until careful roentgenographic examination proves that there is no persistent intrapulmonary infiltration, however small, indicating the presence of such a focus.

Pathological Classification

We have found it helpful to separate the lesions, according to the stage of development, into two groups: simple lung abscess and complicated lung abscess. The characteristics of the two stages are summarized as follows (Figs. 2, 3, 4 and 5):

Simple Lung Abscess	Complicated Lung Abscess
Primary cavity unilocular or multilocular.	Same.
Contents of cavity fluid pus, usually with necrotic slough or semisolid debris.	Same.
Abscess located peripherally in lobe.	Same.
Early pleural symphysis present.	Symphysis usually less extensive if abscess is chronic.
Zone of pneumonitis, varying in width, surrounds primary cavity.	Same, except zone is more extensive.
No daughter abscesses in zone of pneumonitis.	Single or multiple daughter abscesses present in zone of pneumonitis not located superficially and not communicating with the primary cavity.
Secondary bronchiectasis absent.	Secondary bronchiectasis present.
Fibrosis minimal.	Fibrosis minimal to very marked.
Duration: approximately four fifths of cases are under six weeks.	Duration: approximately four fifths of cases are over six weeks.

By correlating the preoperative roentgenographic findings and the observations made during and immediately after operation, it is possible in almost every case to determine whether an abscess falls into the simple or the complicated group. We realize that such a classification is not ideal, inasmuch as it is not possible in many cases to distinguish between a simple multilocular abscess and a complicated one, with its associated daughter abscesses, on roentgenographic appearances alone. In spite of this objection we believe that it is far superior to a purely chronological classification, which gives no consideration to the disease present. To evaluate the results accurately it must first be determined with what one is dealing pathologically. In cases of simple lung abscess all locu-

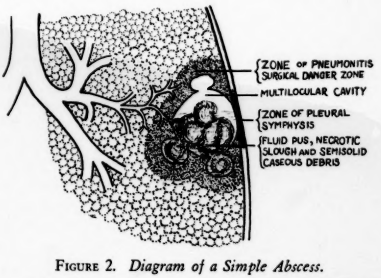


Figure 2. *Diagram of a Simple Abscess.*

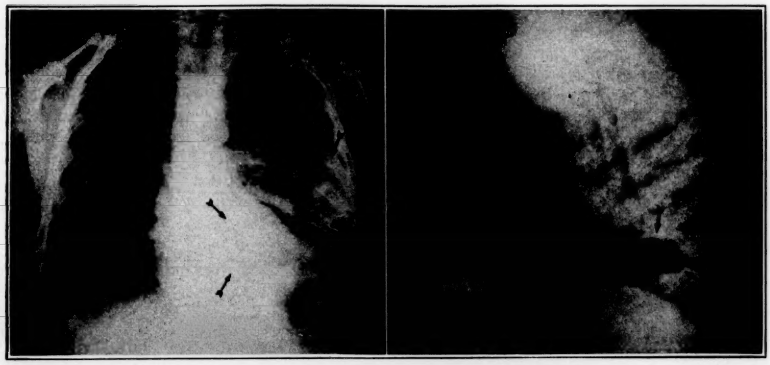
The single abscess cavity is usually multilocular. Semisolid debris within the cavity is a common finding. Note the small caliber of the draining bronchus. Frequently the communicating bronchus becomes partially or completely occluded either with caseous debris from the center of the cavity or from edema of its mucosa.

lations — if present at all — communicate freely with the primary cavity; there is no secondary bronchiectasis, and the amount of fibrosis is minimal. Therefore, one simple incision and drainage of the primary cavity of a complicated abscess can by no means be expected to effect a cure. The persistent isolated daughter abscesses and bronchiectatic dilatations act as reservoirs of smouldering infection, which at any time may flare up into a fulminating pneumonitis or gangrene. When such a course of events follows external drainage of a complicated lung abscess, it is much more probable that it is due to persistence than to recurrence of the disease.

Surgical Danger Zone

Another surgical pathological consideration that deserves special emphasis is that incision, cauterization or any other manipulation of the zone of pneumonitis that surrounds all abscess cavities must be religiously avoided. This zone may very properly be called the "surgical danger zone," because any encroachment on it is attended in certain cases by alarming and even fatal complications. The same warning applies to the traversing of normal lung tissue in an approach to the cavity. Such disturbance breaks down Nature's defense mechanism, which is pitted against virulent and invasive bacteria, thus favoring the development

of a rapidly spreading and frequently fatal pneumonitis. It also invites the next most important group of complications, which are caused by emboli that arise as a direct result of the manipulation. These are manifested clinically by sudden death on the operating table, because of air embolism or other embolism; intracranial suppuration, usually brain abscess or meningitis, owing to the lodgment of septic emboli; other temporary or permanent signs of intracranial involvement such as psychosis or hemiplegia; and generalized pyemia.



A B

FIGURE 3. *Posteroanterior* (A) *and Lateral* (B) *Roentgenograms.*

This was a lung abscess of unknown etiology in a boy of twelve; it had been present for three weeks, with the condition becoming steadily graver. The abscess in the upper lobe developed in the beginning, with rapid spread of infection to the lower lobe, and the development of a second cavity there. Note the fluid level in one of the cavities. Separate thoracotomy for each abscess and single-stage drainage resulted in prompt recovery with complete healing.

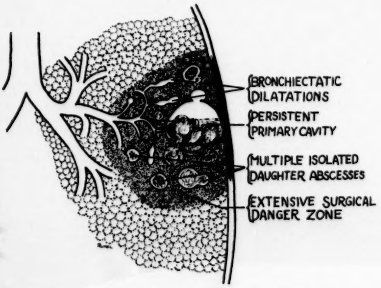


FIGURE 4. *Diagram of a Complicated Abscess.*

The zone of surrounding pneumonitis is extensive. Daughter, or secondary, abscesses fail to communicate directly with the primary cavity. Bronchiectasis has developed in the corresponding pulmonary segment. Empyema may or may not be present. Note the semisolid caseous debris that is found within the cavities.

The risk involved in traversing the surgical danger zone is well shown by a very critical analysis of our cases. After careful study we separated them into two groups: those in which the surgical danger zone had been traversed, regardless of the reason, and those in which this zone had not been traversed. The results are shown in Table 5. An increase in the mortality rate of 300 per cent should certainly make one exceedingly wary of manipulating the danger zone in any way. It should be pointed out that external drainage of lung abscesses in the complicated stage was by far the most important single factor that forced us to traverse the danger zone. It is technically impossible to drain certain daughter abscesses without cauterizing or incising through this zone. The next most important cause of traversing the danger zone was inaccurate localization in our earlier cases.

TABLE 5. *Results According to Whether or Not the Surgical Danger Zone Was Traversed.*

Surgical Approach	No. of Cases	No. of Deaths	Mortality Rate %
Danger zone not traversed	33	3	9
Danger zone traversed	30	8	27*

*Two late deaths from the disease are not included.

Methods of Treatment and Results

Miscellaneous Therapeutic Measures

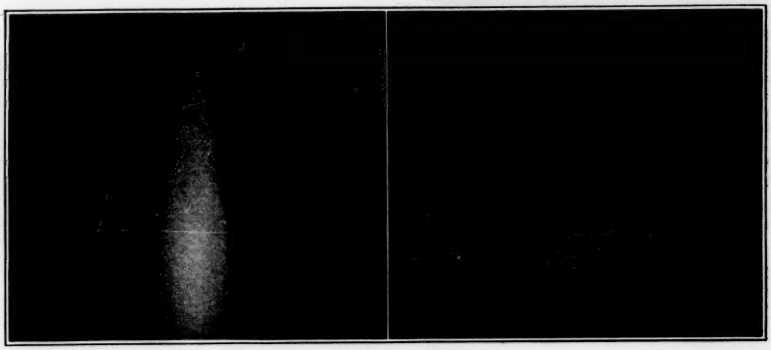
In the earlier cases in our group there was a tendency to employ methods of treatment, especially various types of collapse therapy and supportive measures, that were considered to be more conservative than early external drainage. Our experience with conservative treatment, including both collapse therapy and other measures, has been uniformly unfavorable. Sixteen patients were treated primarily by various types of collapse procedures in different combinations. When one operation failed another was tried. In 11 of the 16 cases after collapse therapy had failed, additional surgery was carried out, usually after the abscess had reached the complicated stage. The results obtained in this entire miscellaneous group, including 3 patients in whom no surgical treatment was given and those treated by collapse therapy, are given in Table 6. The deplorable results speak loudly against this type of treatment. It should be pointed out that the results would be much worse if many of the patients in this group who failed to improve had not finally been treated by external drainage or excision.

Lung Resection

Thirteen patients were treated by excision of pulmonary tissue, 6 by lobectomy, 3 by lobectomy in addition to subtotal excision of an additional lobe, and 4 by total pneumonectomy. The indications for the operations in 11 cases were extensive involvement with fibrosis, multiple isolated abscesses and well-established bronchiectasis demonstrated by lipiodol injection.

In 2 of the 13 cases it was impossible to exclude the possibility of peripheral cancer, and complete excision was deemed the safest procedure. The results are shown in Table 7.

The first postoperative death was due to sepsis following a right middle lobectomy in a man of fifty-five years of age. Excision was chosen because the lesion was suspected of being peripheral carcinoma. The next death occurred in a twenty-four-year-old woman who had had previously a two-stage thoracoplasty that failed to control a post-tonsillectomic lung abscess of the upper lobe. Owing to the previous operations, lobectomy was difficult from a technical standpoint. In addition to this, the lesion had extended from the lower lobe into the upper, which necessitated subtotal



A B

Figure 5. *Posteroanterior* (A) *and Lateral* (B) *Roentgenograms.*

Symptoms of abscess were apparent ten days after herniorrhaphy under difficult general anesthesia in a man of thirty-eight. The original area of pneumonitis and excavation involved only the apex of the lower lobe. Within three weeks there was rapid dissemination of the infection and pulmonary gangrene, with a fatal termination. Note that the suppurative process was limited to the posterior portion of the lung, which was the dependent portion, while the patient remained flat on his back. Note fluid level in lateral film.

Table 6. *Results of Miscellaneous Therapeutic Measures.*•

Results	No. of Cases	Per Cent
Dead	10	53
Living with disease	3	16
Cured	5	26
Unknown	1	5
Total	19	

•At the beginning of treatment there were 10 cases classified as "simple" and 9 as "complicated."

resection of that lobe in addition to the resection of the lower lobe. The patient died four days postoperatively, because of a contralateral bronchopneumonia. The third patient also had a lobectomy with excision of part of an additional lobe for the same reason. Death occurred three days postoperatively from a spreading pneumonitis and cerebral embolism. Lobectomy and pneumonectomy are procedures necessitated only in neglected and complicated cases. If drainage is established during the early simple stage of the disease, resection will seldom be required.

TABLE 7. *Results of Lung Resection.*

OPERATION	NO. OF CASES	OPERATIVE DEATHS	TOO RECENT TO CLASSIFY	CURED
Lobectomy	6	1	0	5
Pneumonectomy	4	0	2*	2
Lobectomy with partial excision of additional lobe	3	2	0	1
Totals	13	3 (23%)	2	8 (62%)

*Asymptomatic but with persistent drainage from empyema.

External Drainage

The success or failure of external drainage of lung abscesses depends on whether or not accurate localization has been established before the incision of the lung abscess is undertaken. This explains the failure of surgeons in the past to treat this disease successfully. They were hopelessly handicapped without the aid of adequate roentgenographic equipment. In spite of the highest degree of surgical skill, they were doomed to failure without exact methods of localization. An error of even an inch, especially in draining simple lung abscesses, frequently spells the difference between a safe incision and one that passes through free pleura or the surgical danger zone or both.

Because of the importance of accurate localization, the details of a method that we have found very helpful will be given.

Localization. The details of procedure are as follows:

Empty the cavity as well as possible by postural drainage immediately before roentgenograms are to be taken. The purpose of this is to allow air to replace fluid, so that the area of excavation may be more apparent.

Take preliminary posteroanterior, lateral and various oblique films, as determined fluoroscopically.

Review the films for preliminary localization.

Inject 2 or 3 minims of a 30 per cent suspension of lamp black in lipiodol (40 per cent iodine) just beneath the fascia of the external intercostal muscle at its junction with the periosteum of the rib that most nearly overlies the center of the abscess cavity. The lipiodol can be seen on the final roentgenograms, and the lamp black shows up clearly at the time of operation. It is therefore possible to be far more exact in approaching the abscess cavity by establishing the relation of the underlying cavity to the suspension as seen in the x-ray films and as seen at operation. A 25-gauge needle with a blunt bevel should be used to minimize the tendency of the suspension to diffuse from the point of injection.

Take final anteroposterior (or posteroanterior), lateral and oblique films—over-exposed if indicated by the preliminary films. The patient should be rotated to localize the abscess properly. We greatly prefer flat roentgenograms with varying degrees of rotation to stereoscopic films.

Operative procedure. The patient is placed in the Trendelenburg position, during operation, to minimize the possibility of cerebral emboli. In most cases the anesthetic of choice is local anesthesia after preliminary medication with Nembutal followed by morphine and scopolamine. This should be supplemented by the administration of oxygen under positive pressure before incision of the pleura, best given by means of a standard anesthesia machine with a tightly fitting face mask. The danger of bronchogenic spread during the operation is thus minimized by preserving the cough reflex and directing the flow of air toward the periphery of the lung. Positive pressure also prevents the lung from falling away from the chest wall if the pleura is opened either intentionally or accidentally.

Surgical technic. A general outline of the operative technic used at the present time is as follows (Figs. 6 and 7):

The thoracic cage is exposed over the site of the abscess, regardless of its position, and the incision is planned to strike the center of the abscess rather than its most dependent point.

The previously injected lipiodol and pigment are then located, as a guide for determining the position of the underlying abscess.

An 8-cm. to 10-cm. segment of the rib over-

lying the center of the abscess is excised, and the stumps covered with bone wax to prevent infection.

The inferior intercostal neurovascular bundle is ligated proximally and distally, and the superior intercostal nerve crushed to decrease the discomfort of postoperative dressings.

Positive pressure oxygen is applied.

An incision is made through the periosteal rib bed and endothoracic fascia — not through the parietal pleura — by sharp dissection. From here the procedure may follow two courses:

IF A PLEURAL SYMPHYSIS IS FOUND:

Prepare the wound of the chest wall to prevent dissecting infection or cellulitis. This is done by suturing subcutaneous fascia to the periosteal bed and obliterating the muscle planes (Fig. 64).

Insert an aspirating needle (17 gauge)

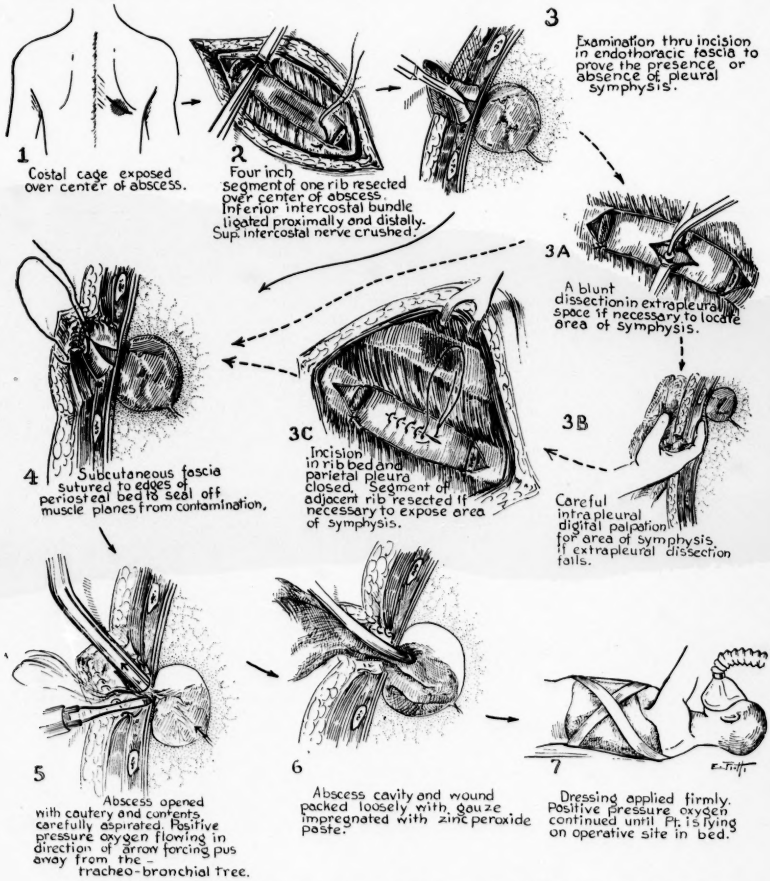
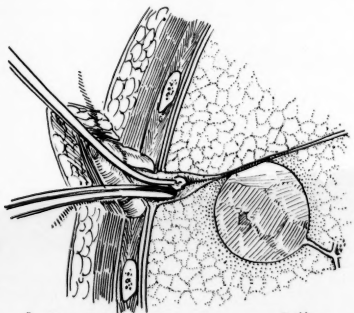

1
Costal cage exposed over center of abscess.
2
Four inch segment of one rib resected over center of abscess. Inferior intercostal bundle ligated proximally and distally. Sup. intercostal nerve crushed.
3
Examination thru incision in endothoracic fascia to prove the presence or absence of pleural symphysis.
3A
A blunt dissection in extrapleural space if necessary to locate area of symphysis.
3B
Careful intrapleural digital palpation for area of symphysis if extrapleural dissection fails.
3C
Incision in rib bed and parietal pleura closed. Segment of adjacent rib resected if necessary to expose area of symphysis.
4
Subcutaneous fascia sutured to edges of periosteal bed to seal off muscle planes from contamination.
5
Abscess opened with cautery and contents carefully aspirated. Positive pressure oxygen flowing in direction of arrow forcing pus away from the tracheo-bronchial tree.
6
Abscess cavity and wound packed loosely with gauze impregnated with zinc peroxide paste.
7
Dressing applied firmly. Positive pressure oxygen continued until Pt. is lying on operative site in bed.

FIGURE 6. *Diagram of Steps in Surgical Drainage of a Pulmonary Abscess That Points on the Costal Cage. 3A, 3B and 3C indicate alternate steps, should one fail to find the area of symphysis at the first attempt.*

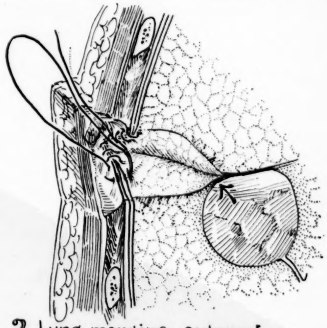
through the area of pleural symphysis and into the cavity. The needle should not be inserted deeply, and repeated attempts to locate the cavity should not be made.

If pus or putrid air is found, leave needle in place as a guide and open the abscess for a distance of 1.0 to 2.5 cm., depending on the cavity size (Fig. 65); aspirate the contents and pack the cavity loosely with gauze impregnated with zinc peroxide paste*; apply boric acid ointment strips to the margins of the wound and dry gauze dressing firmly with positive pressure, continued until the patient has been placed on the operated side.

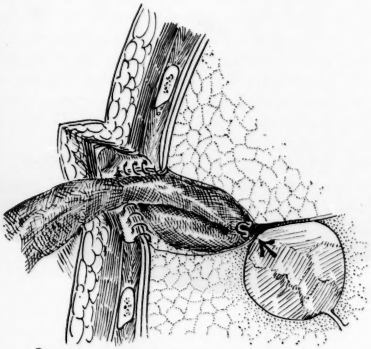
If pus or putrid air is not found, review the localization and, if it is believed certainly that it is correct, cauterize to a maximum depth of 1 cm. over the center of the area of symphysis; if localization is questionable or if no cavity was entered after cauterization, pack with dry gauze with metallic marker in depth of wound; take additional roentgenograms to



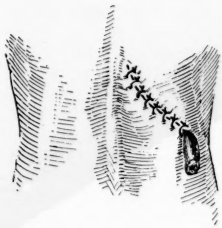
1 Interlobar fissure carefully explored by blunt dissection thru incision in rib bed and parietal pleura to locate area of pleural symphysis.



2 Lung margins sutured to parietal pleura and rib bed.



3 Gauze packed into interlobar fissure with metallic marker against area of pleural symphysis Arrow indicates point at which incision will be made at second stage.



4 Wound closed with interrupted sutures. End of gauze pack protruding from lower angle of wound to allow escape of serum. Second stage incision and drainage carried out in 3-5 days.

FIGURE 7. *Diagram of Steps Used in Preparing Drainage Tract for Lung Abscess Pointing on Interlobar Fissure, Mediastinum or Diaphragm.*

*Recently, sulfathiazole powder has been used instead of zinc peroxide paste. One to 5 gm. is applied to the walls of the lung-abscess cavity and chest wound before the gauze packing is inserted.

check localization and drain the abscess as soon as its position is determined.

If a Pleural Symphysis is Questionable or Absent:

Enlarge the incision and carefully dissect for a short distance in the extrapleural space.

If the adhesions are located in the extrapleural space, reapproximate the edges of the periosteal incision; expose the parietal pleura over the center of the area of pleural symphysis by removing the necessary structures, for example, additional segments of rib or intercostal muscle, and drain through this area.

If pleural symphysis is not found by dissecting in the extrapleural plane, open the pleura, introducing the finger, and carefully palpate in all directions for the point of adherence. If it is found, mark the chest wall over the area of symphysis. Close the pleural and periosteal incision. The drainage tract in the chest wall should then be prepared over the site of adhesions, and the cavity drained. If the precise localization after exploration is uncertain, the wound may be packed with gauze containing a metal clip as a marker, and drainage may be delayed two to four days. During this interval relocalization by roentgenographic study should be done.

If the adhesions cannot be located and the abscess is thought to be pointing on the interlobar fissure, the mediastinum or diaphragm, dissect carefully along the fissure line, mediastinum or diaphragm until the edge of the area of pleural symphysis is approached. Suture the edges of the parietal pleura to the edges of the adjacent lung or diaphragm (Fig. 7). Pack dry gauze with a metallic marker into the depth of the wound against the area of symphysis. Close the wound with interrupted sutures, with the gauze pack protruding from the dependent angle. Take additional roentgenograms for localization. In three to seven days reopen wound and proceed as when pleural symphysis is found.

Results

The results of external drainage of 67 abscesses in 63 patients* are charted in Figure 8. The entire group has been divided into subdivisions; one consisting of 35 patients with simple abscesses and the other of 28 patients with complicated abscesses. The majority of the simple abscesses were under six weeks in duration, whereas the great majority in the complicated group were over six weeks. A comparison of the results in the two groups shows the extreme importance of avoiding delay in carrying out external drainage. All cases

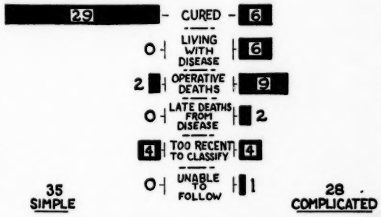


Figure 8. *The Results of Treatment of Pulmonary Abscess by External Drainage, Grouped According to the Extent of Damage to the Lung.*

Four fifths of the simple cases were brought to surgery within the first six weeks of their development. The majority of the complicated cases were late cases.

are included, regardless of the stage of the disease, the general condition of the patient, the previous medical or surgical treatment and other factors. Obviously, if the cases treated early in our experience were excluded and only those treated by the methods now used included, the results would be expected to be much better, owing to the improvement in localization and surgical technic.

One-Stage Versus Two-Stage Operation

The entire plan of procedure used at the present time is based solely on the principle that a lung abscess should be incised only at its most superficial point, so that the zone of pneumonitis will not be traversed in any case and so that aspiration or incision will not be made through a free pleura. Inasmuch as practically all abscesses are located superficially with overlying pleural symphysis, external drainage can be carried out safely in one stage, *provided that the abscess has been accurately localized*. Without accurate localization even a two-stage procedure is a very dangerous plan of management. Although the danger of pleural contamination is theoretically removed, the primary error in localization is not corrected, which means that the subsequent incision must be made through the surgical danger zone. Thus with this plan of treatment the operative mortality rate may be expected to increase by nearly 300 per cent, to say nothing of the high percentage of severe postoperative reactions that will occur.

*Since the above study of results was made, 16 additional cases have come under our observation. Again, we have found that the patient treated during the early acute phase of the disease will recover, whereas delay in surgical drainage spells disaster. Five patients with a simple abscess treated within the first 6 weeks of their illness all recovered. Of the 12 patients treated after the 6-week period 7 recovered. One patient who had suffered from a chronic abscess for 2 years died of an embolism at operation. Eight patients with complicated lesions had had extensive pulmonary destruction. Three patients had extensive gangrene and were moribund when first seen.

Regarding the efficiency with which a two-stage plan of drainage protects against the development of empyema, our results are rather surprising. In the series of 67 abscesses, 24 were drained by means of a two-stage procedure and 43 by a one-stage operation. In the two-stage group, postoperative empyema occurred in 4 cases, whereas in the one-stage group, which is nearly twice as large, postoperative empyema occurred only twice. It should be stated, however, that most of the two-stage operations were done earlier in our experience, when the methods of localization were less exact. Packing the wound for the purpose of creating pleural symphysis is followed at times by an effusion that separates the pleural surfaces. Should this occur the purpose of the two-stage operation is defeated. Furthermore, abscesses have been known to rupture, with the production of empyema before the second stage could be performed.

We believe that the above considerations invalidate any objections to one-stage drainage based on the danger of pleural contamination. The only other objection to a one-stage operation is based on the fear of development of a serious dissecting cellulitis of the chest wall. This objection is theoretically well founded. However, in actual practice the occurrence of such infections is very infrequent. A probable explanation is that the anaerobic organisms rapidly lose their invasive characteristics as soon as they are exposed to the amount of oxygen afforded by a wound that is packed widely open — especially if zinc peroxide paste is used. In the 43 cases with one-stage operations there was only 1 in which a spreading infection occurred, and this cleared up rapidly after a counterincision, 5 cm. in length, was made. Since this experience we have employed measures to obliterate the fascial and muscular planes. This can be accomplished by suturing the subcutaneous fascia to the intercostal muscle bundle. In 27 cases treated in this way spreading infection from the wound edges has not occurred. The danger of cellulitis of the chest wall becomes insignificant when compared with the possibility of pneumonic spread after the first stage of a two-stage procedure. In our group of 24 cases in which two-stage drainage was planned, 2 patients died before the abscess could be drained. A similar experience is commonly mentioned in the literature. We believe that a two-stage operation has a definite and a useful place in the treatment of lung abscess. However, we also consider its usefulness to be limited to a very small group of cases, especially to those in which the abscess is pointing on the mediastinum, on an interlobar fissure without symphysis to the costal cage, and on the diaphragm. It is occasionally necessary when technical difficulties are encountered.

Time of External Drainage

So long as early external drainage continues to produce results similar to those that it has produced to date, we shall consider that, in general, it should be carried out as soon as the diagnosis of lung abscess can be established definitely, and localization can be agreed on. To postpone external drainage is to encourage the relentless progress of the disease from the simple to the complicated stage. This means that the cure rate will drop from 94 to 26 per cent, and that the mortality rate will soar from 6 to 32 per cent (Figure 8). The rapidity with which the disease may pass from the simple stage, through the complicated stage, to death, should be emphasized so that the danger of procrastination may be fully appreciated. The studies of Rives, Major and Romano[12] also indicate the rapid course taken in many cases. In their study of 100 consecutive fatal cases of lung abscess, they found that 73 per cent classed as "frankly hopeless" were of less than one month in duration, and that 75.5 per cent of these cases were less than two months in duration. In view of these figures we are convinced that any feeling for the security of the patient with lung abscess during the first six weeks is false. The frequent statements in the literature that surgery should not be considered until the chronic stage of the disease has been reached, which usually takes from two to three months, are dangerous advice. Associated with such statements is the implication that few if any deaths will occur during this period. When this fallacy is generally comprehended, there should be much less tendency to experiment with uncertain methods of treatment.

We believe that external drainage should be performed without delay for another important reason. We fear that we shall be enticed into the dangerous game of procrastination by the intangible and irresistible human forces far past the point at which our better judgment would ordinarily tell us to stop. This fact is best illustrated by the figures of Brunn,[9] which give the time that surgical treatment was instituted in a group of 205 cases from four institutions, three of which were teaching hospitals in San Francisco. The duration of the disease averaged four hundred and sixty days. The average duration of medical observation and treatment was one hundred and fifty-four days — twenty-eight days short of half a year!

Practically all writers on the subject have stated repeatedly that recovery from lung abscess by conservative means can rarely be expected after the duration has reached three or, at the most, four months. Still, in this group, the average duration of the disease before operation was over fifteen months, and the average duration of medical treatment in the hospitals was over five months. It is interesting to correlate the effect of this delay on the results obtained in this group (Table 2).

Surgical Versus Medical Treatment

A very close analogy can be drawn between the history of the treatment of lung abscess to date and the early history of the treatment of appendicitis. Shortly before the turn of this century, acute appendicitis was considered by the authorities of the time to be a medical disease. The mortality and morbidity rates were high. Laparotomy at that period was feared, largely because of the results associated with this procedure during the time when its various technical obstacles were being surmounted. Therefore, conservative treatment was routinely carried out until the disease had progressed to such an advanced stage that radical surgical aid was asked for, of necessity. For reasons that are now perfectly obvious, the mortality and morbidity rates with surgical treatment were also very high. Because of this, physicians clung even more tenaciously to conservative medical treatment. Thus a vicious circle was formed, and the results of all types of treatment of appendicitis were poor. And they remained poor until such pioneers as J. B. Murphy, Reginald Fitz, J. B. Deaver and others, after a bitter and prolonged struggle against the authorities of the time, showed that, to obtain satisfactory results, surgical treatment must be instituted at an early stage of the disease, before the pernicious forces of the vicious circle are set in action.

At the present time lung abscess is generally considered to be a medical disease. The mortality and morbidity rates are high. Early external drainage is looked on with apprehension. It is feared that some patients will be submitted to unnecessary surgery. We believe that the foundation of this fear has several components, the main one being the unfavorable results obtained by this procedure without exact preoperative localization of the abscess. Because of the general fear of surgical treatment the so-called "conservative measures" are routinely carried out until they are proved to be inadequate, and the patient's life is threatened. Surgical aid is applied after irreparable damage to the lung has occurred, and the results are necessarily poor. Because of this, the general tendency is to cling even more tenaciously to the temporizing medical measures. Again a vicious circle has been formed, which, like a whirlpool, continues to take a high toll in life and health from the unfortunate victims who drift into its irresistible influence.

We firmly believe that the only way to obtain a low mortality rate and a high cure rate in the treatment of lung abscess is to employ early external drainage after accurate localization. Inasmuch as lung abscess is a relatively uncommon disease, we are very much concerned about how long it will take to break the vicious circle that now exists and bring early external drainage into general acceptance. Even though appendicitis was and is a very common disease, it took many years and countless debates at medical gatherings to change the status of its treatment from being primarily medical to primarily surgical. Let us hope, for the sake of the welfare of all future patients who suffer from pulmonary abscess, that the present battle that is in progress in regard to proper management will be soon settled. The problem resolves itself to one of numbers and facts.

Conclusions

The separation of abscesses into acute and chronic stages on a purely chronological basis without regard to the pathologic condition present is unscientific and unsatisfactory. From a practical standpoint it has given rise to a great deal of confusion in evaluating the results of different therapeutic measures. We suggest that this commonly used chronological division be replaced by a classification based on pathology.

It is proposed that lung abscesses be separated into their simple and complicated stages. The simple stage includes a single or a multilocular cavity without secondary bronchiectasis. Complicated abscesses consist of multiple isolated daughter abscesses located in the surrounding zone of pneumonitis, which do not connect with the primary cavity and do not point on the surface of the lobe. In this group we have also included patients who have developed an associated bronchiectasis or empyema.

Lung abscesses are almost invariably situated peripherally with relation to the lobe in which they are located. Therefore, early symphysis between the adjacent pleural layers practically always occurs.

Roentgenographic evidence of cavitation is not essential in establishing the diagnosis. Not infrequently a patient with abscess of the lung will pass through the entire course of the disease to a fatal termination without presenting evidence of definite cavitation.

The generally accepted plan of combined medical and surgical treatment of abscess of the lung continues to keep the mortality over 30 per cent and the cure rate under 50 per cent as shown conclusively by statistics.

In the group of patients treated by us for both simple and complicated lung abscess by miscellaneous methods other than excision or external drainage, the results obtained were poor. The mortality rate was 53 per cent, and the cure rate 26 per cent.

External drainage of complicated lung abscesses has an operative mortality rate of 32 per cent and a cure rate of only 26 per cent.

External drainage of simple lung abscess has an operative mortality rate of 6 per cent with a cure rate of 94 per cent.

The outstanding factor in the development of a complicated from a simple lung abscess is time. Most simple lung abscesses are of less than six weeks' duration. Most complicated lung abscesses are of more than six weeks' duration. Therefore, delay of external drainage, regardless of the reason, parallels directly the bad results obtained. The longer the delay, the worse the results.

Methods of treatment based on internal drainage of the cavity fail because of the small bronchi that lead from it, which provide neither adequate drainage nor adequate aeration of the cavity. These two fundamental factors must be satisfied before healing will occur.

The most important single factor in causing failure of methods based on internal drainage is the presence of semisolid slough or inspissated caseous debris inside the abscess cavities. This precludes the possibility of firm healing and accounts for frequent recurrences.

Intrathoracic spread of the disease is the major cause of death of approximately 85 per cent of patients treated by conservative methods. By far the most important causes of intrathoracic spread are inadequate drainage, faulty aeration of the cavity and any factor that promotes bronchogenic spread of the pus throughout the tracheobronchial tree.

We have outlined in detail the methods of surgical approach, which we believe to be based on sound principles and practical application. The dangers of traversing normal lung tissue or the zone of pneumonitis that surrounds the cavity have been emphasized. With accurate localization one-stage drainage is not only possible but also definitely safer than two-stage drainage in the majority of cases.

Lung abscess should be considered a surgical disease from its onset, and early drainage should be instituted without delay. The provision for external drainage during the simple stage of the disease is the only type of treatment, to date, that has uniformly yielded a high cure rate and a low mortality rate.

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Discussion

Dr. Edward D. Churchill, Boston: A paper on lung abscess always offers a real challenge to those of us who are interested in thoracic surgery, and I am glad to be afforded the opportunity to comment on Dr. Overholt's contribution. Statistical studies on lung abscess at the Massachusetts General Hospital go back to 1909. Dr. Lord in 1925 analyzed the results in 227 cases collected between 1909 and 1924. Two hundred and eleven cases recorded between 1924 and 1932 were reported by Lord and King. This year, Dr. Sweet[13] has published an analysis of 125 cases observed during the five-year period, 1933 to 1937 inclusive. As a result of these studies we have a stable definition of lung abscess for purposes of classification, and unfortunately for our own peace of mind, an exact record of what we have accomplished in the therapy of this disease.

Our best results are recorded in the recent analysis by Sweet of 124 consecutive cases. Sixty-one patients, or 49 per cent, were cured; 42, or 34 per cent, died; and 21, or 17 per cent, remained alive with disease. These figures deal with all cases, early and late, complicated and uncomplicated, ably handled and grossly neglected before entering the hospital.

In the entire group of 124 cases only 8 patients came to the hospital soon enough after onset for drainage to be carried out before the end of the sixth week, so that in general this group is comparable to the "complicated" group of Dr. Overholt's in which he places "four fifths" of his cases that were over six weeks' duration and in which he records a mortality of 32 per cent and a cure rate of 26 per cent.

Now we come to the real challenge offered by this pa-

per. Dr. Overholt states that "external drainage of a simple lung abscess has an operative mortality rate of 6 per cent with a cure rate of 94 per cent." Because "four fifths" of the abscesses classified as "simple" were subjected to external drainage within six weeks of onset the conclusion is drawn that "external drainage should be carried out as soon as the diagnosis of lung abscess can be established." If the figures support this conclusion, a specific remedy has been found for a devastating disease that hitherto has carried a mortality rate of 34 or more per cent. When so important an announcement as this is made, the author invites a very critical survey of his documentary evidence.

The series of 35 cases of "simple" abscesses were apparently selected nonconsecutively from a larger group that includes the "complicated" cases. "Approximately four fifths" of the 35, that is, 28 cases, were subjected to external drainage within six weeks from time of onset. It is to be noted, however, that 5 or 6 other cases (one fifth of the group of 28 "complicated" cases) were also subjected to external drainage within six weeks of the date of onset. They are placed in the "complicated" group, in which the group mortality is given as 32 per cent, but no specific information is given regarding the fate of these 5 or 6 patients, in whom we are intensely interested.

We may therefore conclude that Dr. Overholt has drained lung abscesses of less than six weeks' duration in 34 (28 plus 6) patients. Two patients (6 per cent of 35) in the "simple" group died, and lacking information to the contrary, let us assume that all 6 in the "complicated" group died. This gives a mortality of 8 out of 34 or 23 per cent for cases drained under six weeks.

This line of investigation can be carried further. There is no note of spontaneous cures. During two successive five-year periods our spontaneous cure rate has been 21.4 per cent and 19.3 per cent respectively. If it is assumed that 20 per cent of Dr. Overholt's early abscesses would have subsided spontaneously if allowed to do so, it is permissible to state that 7 cases were operated on as a prophylactic measure and proceeded to get well, as they would have done without operation. Subtracting 7 cases from the total of 34, leaves but 27 cases in which external drainage as a curative measure was necessary. Eight of these patients may have died, as already noted—a mortality rate of 30 per cent. Dr. Sweet has shown in an ingenious table that a patient with an abscess of six weeks' duration faces a mortality rate of 28.9 per cent. A remarkable coincidence!

I know that I have been hypercritical and also quite possibly unfair, particularly in assuming that all 6 patients operated on early and placed in the "complicated" group died. However, it is not sound reasoning to conclude that abscesses should be drained as soon as the diagnosis is made on the basis of results in a selected group of simple abscesses.

So much for the statistical evidence. Another phase of the paper puzzles me a great deal. Within six weeks of onset Dr. Overholt has arrived at a diagnosis of lung abscess and proceeded with external drainage in 34 out of a total of 94 cases during an eight-year period. At the Massachusetts General Hospital the diagnosis has been made and the patient admitted to the hospital under six weeks in but 8 cases out of a total of 124 in a five-year period. I have always thought that in New York Dr. Neuhof might have an unusually alert and well-informed group of medical associates to provide him with his material, but Dr. Overholt brings the problem closer home. Is it possible that we are working with different definitions of lung abscess and that our clinical material is not comparable for this reason? This cannot be judged until Dr. Overholt presents a more detailed account of the patients operated on within six weeks—an account that lists age, sex, location of the abscess, x-ray findings, and particularly etiology.

I find myself in accord with one very important conclusion of Dr. Overholt's paper, a conclusion that should not be overlooked in a squabble about details. The nonoperative treatment of lung abscess in the community as a whole is being continued far beyond the point where any reasonable chance for spontaneous cure has passed. Irreparable damage to the lung results, and when surgery is called for, a more radical procedure than external drainage must be employed.

It is not possible to determine by any rule-of-thumb method just when a reasonable expectation of spontaneous cure ceases. This can be judged in the individual case only by careful clinical and laboratory observation, and as a result of experience in the management of the disease. A sound knowledge of the pathology of lung abscess makes it unnecessary to try this or that newly proposed remedy to know whether or not it will work.

REMOVAL OF IODIZED OIL BY LUMBAR PUNCTURE

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BECAUSE of its irritating effects, there has been an increasing tendency to forego the use of iodized oil in myelography and to employ the surgical condition is present, and the reluctance to use iodized oil under such circumstances is often responsible for prolonged disability of patients

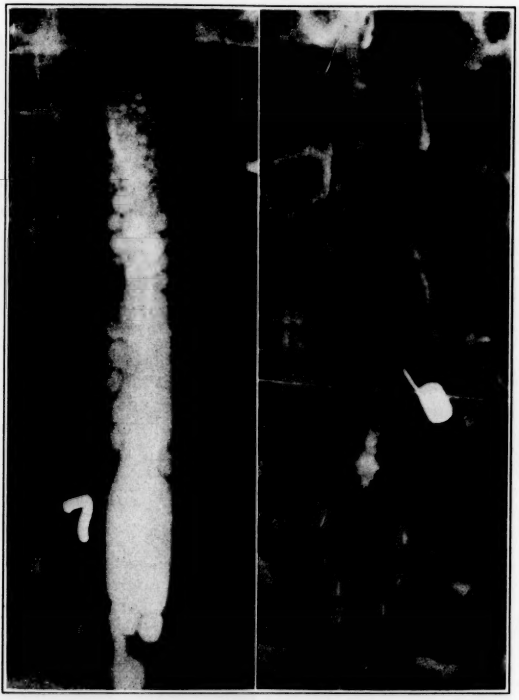

FIGURE 1.

Roentgenograms before (A) *and after* (B) *removal of iodized oil, injected into the lumbar subarachnoid space four days before removal. Small amounts of oil remain in the cul-de-sac and in the sheaths of the nerve roots. The dense shadow represents the hilt of the lumbar-puncture needle, inserted between the third and fourth lumbar vertebrae; the heavy transverse line, a needle on the surface used as a marker.*

less reliable procedure of pneumomyelography. If the oil can be removed shortly after its introduction, as may be done when it is accessible at operation, there is no serious objection to its use. One cannot always be certain, however, that a who might be relieved by surgery. This is particularly true when a ruptured lumbar intervertebral disk is suspected but the history and findings are inconclusive. By the use of iodized oil it can be determined, almost without exception, whether or not a ruptured disk is present, whereas a pneumomyelogram still leaves one in doubt.

We are, therefore, submitting a simple method

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for the removal of iodized oil from the lumbar subarachnoid space.

After the roentgenographic examination has been completed, the patient is placed face down, horizontally, on a fluoroscopic table; the iodized oil is visualized; with the patient still in the prone position, a lumbar puncture is made in the space beneath which the oil is located. The oil, or the greater part of it, is then withdrawn by gentle suction with a small syringe. By tilting the table, under fluoroscopic control, any remaining oil is brought to the point of the needle and removed. Since small droplets of the oil may not be visualized with the fluoroscope, a film is taken before the needle is withdrawn, to make sure that removal of the oil has been as complete as possible.

A few comments may be added. Before performing the lumbar puncture the table is tilted 20° or so, raising it at the head end, to distend the lumbar cul-de-sac. Then the table is leveled again, bringing the oil back to its original position, or centered round the needle, the point of which should be in the ventral portion of the subarachnoid space. If suction is too strong, pain may result and the flow of oil may stop, presumably because a nerve root or fold of membrane has been drawn against the point of the needle. Using less force and turning the needle overcome this difficulty, which is not very serious and is more likely to occur when the needle, in being inserted, has deviated too far from the mid-line. An ordinary 18-gauge lumbar-puncture needle is entirely satisfactory; oil introduced only a few hours previously and still very nearly in its original, thick, viscid state can be removed through it without difficulty. When, on the day following introduction or later, the oil is emulsified, it flows more easily, but if it has entered the sheaths of the nerve roots, some of it may not be recovered. We believe that a few globules of oil left free in the lumbar cul-de-sac are of little or no importance, although with a little persistence all of it can be removed. Needless to say, the discomfort and incapacity suffered by the patient are insignificant.

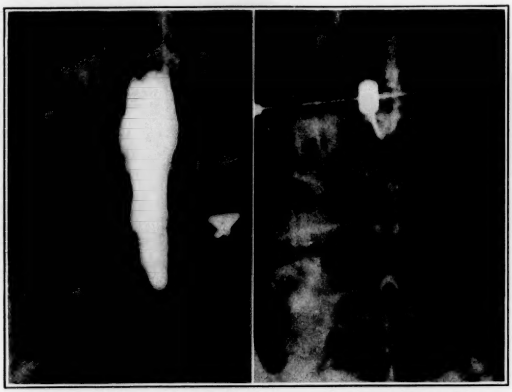

FIGURE 2.

Roentgenograms before (A) *and after* (B) *removal of iodized oil, injected five hours before removal. A few droplets of oil remain in the cul-de-sac. The lumbar-puncture needle was inserted between the third and fourth lumbar vertebrae.*

Since the above procedure results in more complete removal than is usually possible at operation, it may be used with advantage, if the oil is accessible, even when there is to be an operation later. Another reason for preoperative removal in cases of ruptured lumbar intervertebral disk is that it obviates incision of the meninges, with resulting loss of spinal fluid and unnecessary exposure of the subarachnoid space to infection. The method may also be employed in suitable cases of suspected compression of the cervical or thoracic cord by introducing the oil into the lumbar subarachnoid space instead of the cisterna magna.

We have used this procedure in over 30 cases. It is most effective when the needle is inserted through the third or fourth lumbar interspinous space, although complete removal has been ac-

complished through the second and, on one occasion, the first lumbar space. One obviously tries to avoid the suspected lesion.

It is important to make a neat puncture so that the point of the needle will be near the mid-line, to use no more suction than is necessary to bring the oil very slowly up into the syringe, and not to continue or, what is worse, increase suction if the needle is blocked. The needle in Figure 1 is badly placed.

For some time we have been introducing the oil with the patient on the fluoroscopic table, leaving the needle in place during the examination, and removing the oil immediately afterward.

THE AUTONOMIC NERVOUS SYSTEM PRIOR TO GASKELL*

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IN a charming tribute to his former teacher, Langdon-Brown[1] (1939) writes provocatively that "to read an account of this [autonomic nervous] system before Gaskell is like reading an account of the circulation before Harvey." During a recent search through the writings of physiologists just prior to Gaskell and Langley, I had the opportunity to ponder this statement and to wonder why so many threads of evidence lay unrecognized until these two men, so curiously different in temperament, began to weave them into the pattern of the visceral nervous system as we know it today. The osmic acid technic, then recently introduced by Schultze, no doubt enabled Gaskell to see the fibers of the autonomic nerves in much clearer outline, but one should not attach too much importance to this factor, since the Weigert method of staining myelinated sheaths had often been used before in studying the constitution of the ventral spinal nerves and, indeed, of the rami communicantes. Gaskell[2] built his theory on two main lines of evidence: that only the ventral roots in the thoracolumbar and sacral regions contained fine myelinated fibers, and that these fibers were present only in the white rami that were limited to the same regions.‡ It is interesting that, although true as generalizations, both observations proved to be inaccurate in the light of subsequent investigation. Nevertheless they gave Gaskell the clue to a new understanding of the construction of the autonomic nervous system. The difference between the thoracoventral roots and those elsewhere had been noted as early as 1862 by Reissner,[3] and it must have been seen by many others, undoubtedly by the great Henle[4] and by Kölliker,[5] without their appreciation of its underlying significance. There can be little doubt that Gaskell possessed a quality of genius that enabled him not only to see but also to comprehend.

In like manner, the effect of nicotine in blocking autonomic responses, described first by Hirschmann,[6] gave to Langley another new weapon with which to attack an age-old problem, but one must not overlook the fact that it was Langley[7] who, in collaboration with Dickinson, showed that nicotine acts by interrupting the synapse in the sympathetic ganglion, a fact that was fundamental in Langley's approach. Nor can one deny that Langley's genius demanded of him years of incessant and meticulous search, which led to achievements that, in the words of Fletcher[8] "stand permanently in their places not merely as additions here and there to knowledge, but as indispensable stepping stones along which, at this point or that, the progress of knowledge has actually made its way."

In looking for the germ of any modern idea, one is tempted to turn at once to Claude Bernard,[9] who so often predicted the discoveries of the present day. Prior to Bernard, views on the "vegetative" nervous system were characterized by a wealth of theoretical argument and a poverty of factual knowledge. To be sure, the cerebral origin of the sympathetic "intercostal" nerve had been disproved in 1727 by Petit,[10] and the fine caliber of its constituent fibers had been ably demonstrated in 1838 by Remak,[11] who described the unmyelinated fibers, and in 1842 by Bidder and Volkmann,[12] who first defined the fine medullated fibers as sympathetic. But the rami communicantes were little understood, except as connections between the cerebrospinal nervous system and the independent vegetative nervous system of Bichat, and there had been much discussion as to whether they communicated with the ventral or dorsal spi-

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‡Gaskell apparently counted fifteen, instead of thirteen, thoracic nerves in the dog, so that the subsequent lumbar and sacral nerves in men and dogs might have the same numbering. Thus he gave as the lower limit of the thoracolumbar outflow in the dog, the twenty-fifth spinal nerve, which he called L2, instead of L4 as it is customarily designated. This rather unfortunate method of numbering led to considerable confusion in subsequent interpretation.

nal roots or both (Müller,[13] Retzius,[14] Wützer[15]). Beck,[16] in 1846, was probably the first to point out that the white rami consist of myelinated (tubular) fibers that emerge from the spinal cord, and that the grey rami contain unmyelinated (gelatinous) fibers arising within sympathetic ganglia, probably in connection with the cells described earlier by Ehrenberg.[17] I[18] have already reviewed this early phase in the literature, and the following remarks on the period just prior to Gaskell should be considered as supplementary to that paper.

It was apparently Senac[19] who, in 1794, first discussed the contractility of the blood vessels in relation to the sympathetic nerves, which he described as embracing the larger arteries and sending fibers along their finest ramifications. Certainly Petit[10] in 1727 and subsequently Cruikshanks[20] (1795), Arnemann[21] (1797) and Reid[22] (1838-39) had noticed a redness of the conjunctiva after section of the cervical sympathetic ganglion, but they did not appreciate the significance of their observation, nor did Dupuy[23] (1816) and Brachet[24] (1837), who described the same "inflammation" of the conjunctiva and an elevation of skin temperature on the ipsilateral side of the face after extirpation of the superior cervical ganglion. Brachet came nearest to the present concept of vasomotor tonus when he remarked that the "inflammation" was not an excitatory effect but an atony or paralysis. Then came the irrefutable demonstration by Henle,[4] in 1840, of muscular elements in the walls of blood vessels. Before the work of this histologist, the middle coat of the arteries had been designated by different authors as the "muscular tunic," but the term had been purely visionary. Physiologists who had upheld the contractility of vessels had to suppose the presence of muscle in their walls and by happy inspiration had placed it in the tissues of the middle coat. Henle's description and its confirmation in 1849 by Kölliker[5] removed all doubt about both the arteries and the veins, and the authors did not hesitate to attribute to the nervous system an action on the muscular wall of the vessels. Although experimental proof was lacking, the idea must certainly have been in the minds of physiologists during the first half of the nineteenth century, for in 1840 Stilling[25] proposed the substitution of the term "vasomotor system" for the name "sympathetic." The ground was well prepared for the experimental work of Schiff,[26] Claude Bernard[9] and Brown-Séquard[27] that provided the proof for the existence of vasomotor nerves. The story of this is so ably told elsewhere (Fulton[28]) that it need not be dwelt on any further.

The demonstration that such vasomotor fibers emerge from the spinal cord followed logically Budge and Waller's[29] account in 1851 of the pupillary fibers in the lower cervical and upper thoracic regions of the cord and passing out in the cervical sympathetic.* "One of the great merits of the experiment of Budge and Waller," wrote Bernard,[9] "is that it showed for the first time in an incontestable way that the fibres of the great sympathetic take origin from the spinal cord." And following this line of reasoning, Bernard cut the ventral and dorsal roots of all the nerves that supply fibers to the brachial or the lumbosacral plexus, without causing any change in vascularity or temperature in the corresponding paralyzed and anesthetic extremity. It mattered little whether the ventral or dorsal roots or both were divided. Therefore, concluded Bernard, the vasomotor nerves to the extremity do not arise in the spinal roots that furnish the motor and sensory fibers to the limbs. When, however, he divided the nerve trunks of the plexuses (brachial or lumbosacral), there appeared in addition to the motor and sensory loss an immediate rise in temperature of several degrees in the corresponding foot. This proved that the vasomotor fibers join the motor and sensory nerves to the extremity outside the vertebral canal, and Bernard's attention was naturally attracted to the sympathetic chain. The next logical step was the removal of the stellate ganglion, or of the lumbar sympathetic chain lying on the bodies of the fifth and sixth lumbar vertebrae, and Bernard then obtained the rise in temperature without the motor or sensory changes. Stimulation of the sympathetic chain had the opposite effect of reducing the circulation and lowering the temperature in the limb. The proof of independent vasomotor fibers was therefore complete, but, curiously enough, Bernard did not push the matter further, and the tracing of the exact outflow of the fibers from the cord was left to others. In a significant passage, however, he comments:

> It seems to me that sometimes the temperature changes in the posterior extremity followed stimulation of the regions of the spinal cord higher up [than the origin of the lumbosacral plexus], and I have seen an increase in temperature in the anterior extremity and in the ear on cutting the sympathetic chain at the level of the third and fourth intercostals, without any accompanying ocular phenomena.

Thus Bernard predicted that the outflow of the vasomotor fibers would be found in the region of

*Budge[30] relates that Petit made his first experiment in Namur in 1712, and later (in 1725) repeated it before Winslow, Senac and Hunant in Paris. Petit had demonstrated myosis on section of the cervical sympathetic, but Biffi,[31] in 1846, was the first to show that stimulation of this nerve caused dilatation of the pupil. The experiment was repeated by Waller in Bonn, where Budge was then working; thus in 1851 the collaboration of Waller and Budge on the innervation of the iris began.

the spinal cord between the cervical and lumbar enlargements.

The account of the vasomotor nervous system written by Vulpian[32] about the time of Bernard's death, and more than a decade before Gaskell's classic paper, reveals an extraordinarily modern outlook. The rami communicantes were by this time fully understood. They were composed of two groups of fibers, one traveling from the spinal cord chiefly along the ventral roots, the other arising in the ganglia and running to the periphery. The vasomotor nerves along their course from the cord to the blood vessels were therefore believed to communicate with one or more sympathetic ganglia. In 1877–1878 Luchsinger,[33] having confirmed Goltz's[34] observation that stimulation of the sciatic nerve caused sweating on the corresponding foot, established the existence of true sudomotor fibers in the sympathetic nerves, and proved by stimulation experiments in the cat their emergence from the spinal cord, to the leg from T10 to L4 inclusive and to the arm from T3 to T6 inclusive. Thus the physiologists began placing first the vasomotor, then the sudomotor and ultimately the pilomotor fibers within the thoracolumbar outflow as we know it today, and as early as 1884 Dastre and Morat[35] wrote: "From the anatomical standpoint there is a remarkable arrangement, for the sympathetic appears to arise principally from the thoracic region." The authors added, "One can assign to the sympathetic centers a distinct position in the cord, in a chain of ganglionic tissue occupying the outer angle of the anterior horn and known as the tractus intermedio-lateralis."

In like manner, Bernard,[9] Schiff[26] and Vulpian[32] had shown the existence of vasodilators in certain of the cranial nerves, — the chorda tympani and glossopharyngeal nerves in particular, — and Eckhard[36] in 1863 had demonstrated a sacral outflow of vasodilators in the nervi erigentes. The relation of the vagus nerve to the "organic" nervous system had been established in 1846 when the Webers[37] showed its inhibitory cardiac effect, and the cranial ganglia, the ciliary, sphenopalatine, submaxillary and otic, had all been described and regarded as belonging to the ganglionic nervous system by Arnold[38] in 1827. Meanwhile the embryologic studies of Schenk and Birdsall,[39] of Balfour,[40] and particularly of Onodi[41] had given final proof of the spinal origin of the sympathetic trunk.

Thus, as in Bernard's discovery of the vasomotor nerves, the ground was well prepared for Gaskell. This in no way belittles the greatness of his contribution, for we have but to turn to a work similar to his that appeared two years earlier to see how differently the same data could be interpreted. Schwalbe[42] had been interested in a proposition that the diameter of a nerve fiber might vary directly with its length, and he therefore examined the ventral roots of several cervical and thoracic nerves in man. Observing that the cervical roots contained only a few fine fibers, whereas in the thoracic roots there were numerous bundles of fibers of 1.8 to 2.7 microns in diameter, Schwalbe unfortunately argued that the predominance of small fibers in the thoracic nerves was owing to the fact that they had a shorter course to the periphery than the cervical nerves extending into the arm. Indeed it was some years before the magnitude of Gaskell's advance was appreciated, even by those working in the same field, as is evident from Kölliker's involved account of the sympathetic nervous system in 1894.

To Gaskell, then, must be given the credit for the discovery of the thoracolumbar and craniosacral outflows, although perhaps the story as related here may not fully substantiate the commentary of Langdon-Brown, quoted at the beginning of this article. But to quote again from his delightful paper, "the splendid accuracy of Langley's mental microscope extended and completed the discoveries made by the great sweep of Gaskell's mental telescope," and in illustration of this I call attention to that extraordinarily prophetic statement of Gaskell's[2] written in 1886:

> The evidence is becoming daily stronger that every tissue is innervated by two sets of nerve fibres of opposite characters so that I look forward hopefully to the time when the whole nervous system shall be mapped out into two great districts of which the function of the one is katabolic, of the other anabolic, to the peripheral tissues: two great divisions of the nervous system which are occupied with chemical changes of a synthetical and analytical character respectively, which therefore in their action must show the characteristic signs of such opposite chemical processes.

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SUSCEPTIBILITY TO AND LATENCY OF POISON-IVY DERMATITIS*

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SINCE there is considerable difference of opinion about the individual susceptibility to and the period of latency of poison-ivy (*Rhus radicans*, L.) dermatitis, we carried out a series of tests on a group of 23 volunteers.

Knowles et al.[1] found their percentage of reactors to the patch test (50 per cent in 1938 and 40 per cent in 1937) definitely less than that reported by Spain[2] (65 per cent), by Deibert and his associates[3] (59 per cent) and by Straus[4] (76 per cent). They observed a quicker response to the leaf itself than to any of the solutions employed. The period of latency in their 56 patients tested with the leaf varied from twelve hours to over one hundred and twenty.

McNair[5] states:

> Fontana [in 1795] found the period of latency to be three, four and six days. Van Mons, in 1797, found it to be from eight to ten days; Horsfield, in 1798, stated that the period of latency varied from a few hours to several days . . . Dakin (1829), one to three days; Busey (1873), seven days; White (1873), generally less than forty-eight hours but sometimes five days; Park (1879), from a few hours to a few days, generally from thirty-six to forty-eight hours; Cundell-Juler (1883), about twenty-four hours; Morrow (1887), from several hours to four or more days; Pfaff (1897), . . . as long as four days; Conner (1907), often within twenty-four hours.

In 18 cases, McNair noted that the period of latency varied from one to eight days.

A poison-ivy plant was potted and maintained fresh for the experiment. All the original tests were carried out in the early spring within an interval of two weeks. A fresh and previously untouched leaf was removed from the plant at the time of each experiment. A section 2 cm. square was cut from the leaf and rubbed immediately with moderate friction for approximately thirty seconds on two skin areas, one on the flexor surface of the forearm and the other on the lower leg. A dry dressing was applied, and the subject

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was instructed not to wash the areas inoculated until permission was granted. Daily observation was made of the inoculated areas.

Of 3 subjects who presented no reaction at the end of one month, 2 were retested. The same method was repeated, except that the leaf was rubbed energetically on the skin — in an area previously untouched — until bleeding points had formed, after which small bits of the leaf were strapped to the skin areas for twenty-four hours.

The persons thus tested were of both sexes and were all between twenty and thirty years of age. There were approximately equal numbers of Anglo-Saxons, Italians, Irish, Armenians and Jews. The skins of all those tested were neither excessively dry nor seborrheic.

TABLE 1. *Results.*

Case No.	Incubation	Type of Reaction*	Duration
	days		*days*
1	2	Erythema; papules; pruritus	7
2	18	Small papules; pruritus	2
3		No reaction†	
4	11	Erythema; no pruritus	14
5	6	Fine papules; pruritus	10
6	1	Erythema; vesicles; pruritus; entire flexor surface of arms and forearms affected	21
7		No reaction†	
8	2	Erythema; vesicles; entire flexor surface of arms and forearms affected	15
9	2	Erythema; vesicles; entire flexor surface of arms and forearms affected	21
10	2	Vesicles	21
11	10	Erythema; edema; vesicles	21
12‡	3	Erythema; edema; vesicles; generalized macular eruption	14
13	2	Erythema; edema; bullae	14
14	11	Vesicles	2
15‡	5	Erythema; edema; vesicles	14
16	11	Papules	5
17	4	Papules; vesicles	11
18‡	1	Vesicles	5
19	5	Erythema; edema; papules; vesicles	8
20	3	Erythema; vesicles	10
21	2	Vesicles	12
22	8	Papules	3
23		No reaction	

*All reactions limited to the areas inoculated, unless otherwise noted.
†Reinoculated, with similar result.
‡History of previous attacks of poison-ivy dermatitis.

The results were as follows (Table 1). Twenty of the 23 subjects gave a positive reaction. Of these, 15 reacted to the first known exposure. The shortest period of latency was one day, the longest eighteen days and the average six days. The duration of the reaction varied from two to twenty-one days. The cases of shortest incubation and most marked reaction were usually those of longest duration as well.

There were no apparent variations according to sex, race or site of inoculation.

Whereas one of the darker-skinned subjects (Case 4), had a long period of latency and a very mild reaction another (Case 9) had a short period of latency and a marked reaction.

The two subjects who showed immunity, even after the second test, were Jewish, and fair of skin. One perspired profusely, with an otherwise normal skin; the other had a skin in no way different from the other subjects tested. Neither had ever experienced an attack of poison-ivy dermatitis.

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CLINICAL NOTE

A SIMPLE MICROMETHOD FOR THE DETECTION OF ACETONE IN URINE

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THE usual tests for acetone in the urine are the Legal test[1] and such modifications as those of Jackson-Taylor,[1] Lange[2] and Rothera.[2] A modification[3] of the Rothera test employs a powder derived from a mixture of ammonium sulfate and sodium nitroprusside. All these tests require several cubic centimeters of urine and have sodium nitroprusside as their principal reagent. In the test to be described, only a few drops of urine are required, and the entire procedure is simplified.

The principal reagent for this test is a 5-gr. (0.35-gm.) tablet† made from a mixture of 1 part of finely powdered sodium nitroprusside and 40 parts of ammonium sulfate. The tablets are stable.

For the test, a tablet is placed on a white porcelain tile or dish. Three drops of urine are then placed on the tablet, followed by two drops of concentrated ammonium hydroxide. If acetone is present, a purplish color appears at the periphery of the tablet within thirty to sixty seconds, and becomes maximum within three to five minutes. After five minutes, the color tends to change to brown. For this reason, the test should be read at the end of five minutes.

In comparing this test with the usual macromethods, predetermined amounts of acetone were added to normal urine, and the tests were done simultaneously. In the higher concentrations of acetone, there was complete agreement of the two methods, but when the concentration of acetone became low, the micromethod was noted to be slightly more sensitive. For example, when the concentration of acetone in the urine was 0.02 per cent, a clearly positive reaction was noted with the micromethod, whereas the reaction was equivocal with the macromethod.

This test is an improvement on the macromethod because it is simpler and involves fewer steps and less apparatus; it can be performed on very small amounts of urine—3 to 4 drops instead of 3 to 4 cc.; it is timesaving, because many such urine tests can be set up and done simultaneously; it is more economical because it saves on reagents; it is as accurate as the macromethod and slightly more sensitive with low dilutions of acetone.

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MEDICAL PROGRESS

POPULAR MISCONCEPTIONS PERTAINING TO OPHTHALMOLOGY

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PROGRESS consists not alone in the appreciation and utilization of newly discovered facts but also in the realization and correction of prevalent misconceptions. Although the majority of misconceptions pertaining to ophthalmology are not serious in the sense that they interfere with medical practice, they are, in view of their universality, the source of unnecessary health precautions and of a not inconsiderable economic waste. So far as health is involved, it is the privilege and obligation of the medical profession, presumably the most qualified judge, to exercise its authority.

ILLUMINATION

The beliefs that relatively intense illumination is necessary for visual efficiency and that weak illumination induces organic disease of the eyes are probably the most widely held misconceptions pertaining to ophthalmology. This is represented in the following quotation from a semiscientific treatise: "Improper lighting conditions and the misuse of light and the eyes are so effective in causing eyestrain and permanent injury to the eyes."[1]

The public has been subjected to a "research offensive"[2] in matters of illumination, aimed toward the development of a widespread consciousness of lighting. As a result, there has been a "newborn confidence [that] increases interest and decreases resistance on the part of the user of light and lighting."[3] The nucleus of this movement is in the Incandescent Lamp Department and the Lighting Research Laboratory of the General Electric Company from which have come an impres-

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sive number of technical and popular books and pamphlets by Luckiesh, Moss and their collaborators. These authors claim to have established a new science "born of radically new concepts,"[2] the science of seeing. This science transcends the usual ophthalmologic and optometric practices, which "seek primarily to correct ocular defects rather than to prevent them."[4] According to this new creed, the optimal amount of artificial light is said to be far in excess of that now prevailing in "a half seeing world,"[5] and thanks to the researches of this science, the "deeply hidden effects of seeing which are unnecessarily wasting human resources are at last revealed."[6]

The foundation of this science of seeing is both philosophic and experimental. Philosophically, it is based on the assumptions that man evolved in sunlight and that the aim of artificial lighting should be to produce indoor illumination intensities "competing with daylight."[7] Although the increase in lighting intensities necessary to produce these allegedly ideal conditions amounting to thousands of foot-candles are as yet impractical,[8] the sponsors of these ideas advocate a compromise — one that, they assert, further technical advances in lighting may make temporary only. Needless to say, the recommendations of this science, even with the compromise allowance, have come as a considerable surprise to those familiar with man's ability to adapt himself to dark, and to those who are acquainted with previous reports showing that 10 foot-candles are adequate for ordinary purposes, and not more than 20 foot-candles for exceptionally fine work.[9, 10]

The experimental data that, according to Luckiesh and Moss, have given rise to the "revolutionary"[11] concepts of the science of seeing are derived from two sources: first, from observations with the Luckiesh–Moss visibility meter, which allegedly indicates in foot-candles the intensity of light necessary for any visual task; and secondly, from a number of biologic changes that the authors claim to have correlated with ease of seeing.

The measurements with the visibility meter consist in interposing between observer and test object a variable filter. The test consists in resolving two vertical bars while the total illumination reaching the eye is varied by means of the filter.[12] Of course, the same result, so far as illumination is concerned, might be obtained simply by varying the intensity of light on the test object. The visibility meter, however, does something else: it introduces another variable through the progressively increasing scatter or fogging effect of the filter. To overcome this combined result of reduction in intensity and increase in fogging, the authors quite naturally find that high degrees of illumination on the test object are necessary. They are, in fact, led to conclude that intensities of 100 foot-candles are desirable for ordinary reading.[13, 14] Wholly aside from the fact that the artificial conditions set up have no common counterpart in Nature, the authors have used visual acuity as a measure of visibility, a criterion that has been uniformly shown by many investigators, although not mentioned by Luckiesh and Moss, to reach a maximum at about 10 foot-candles.[15] It is somewhat astonishing that these authors, who contend that it is "necessary to reveal the shortcomings of loose statements or dogmatic opinions,"[16] should show such a patent disregard for the relevant though contradictory experiments of others. Because of the unnatural conditions set up by the visibility meter and because its recommendations are contradictory[17] to almost all previous pertinent investigations, it cannot be accepted as a valid test without other convincing evidence.

The other source of experimental data that is said to prove the necessity of light intensities considerably in excess of present conditions is the alleged correlation of different illuminations for reading with certain biologic changes. Specifically, the authors state that reading under low illumination produces increased tension of the finger on a push button,[18] increased size of pupil,[19] slowing of heart,[20] decrease in amplitude of convergence,[21] increased frequency of blinking,[22] and more recently, changes in the rate of reading.[23] Some of the data have been misinterpreted by the authors,[24] and some have been contradicted by the work of others.[25] None of the experiments are above controversy, and all are done with the wide intensity variations of 1, 10 and 100 foot-candles, — in some experiments 0.1 and 10 foot-candles only were used, whereas in others only 1 and 100 foot-candles, — so that deductions concerning the intermediate intensities, which are the significant ones, of 5, 10, 20 and 40 foot-candles are invalid. The criteria of comfort and work output are, according to the authors, unreliable. Thus they dispose of the considerable amount of data of previous investigators that indicate that for all practical purposes light intensities of 10 foot-candles are adequate.[9, 26–29]

Sweeping conclusions based on the above experiments form the substance of most of the literature that during the last fifteen years has inundated engineering, psychologic, industrial, medical and popular journals recommending ever-increasing illuminations. At the same time, a considerable number of reports, less accessible to the public perhaps, but nonetheless authoritative, have led

to the conclusion that 10 foot-candles is sufficient illumination.[30–35] It is pointed out in these latter reports that visual acuity increases rapidly from 1 to 5 foot-candles, slowly from 5 to 10 and only slightly above 10. Certainly intensities greater than 20 foot-candles have no practical significance.[15]

But from the medical point of view the unfortunate part of this higher illumination campaign lies not alone in the fact that it is unjustified on the grounds of efficiency. What is worse is the implication that the illumination in the average home and office results in some organic harm to the eyes, and thus the public is cajoled into increasing the lighting for presumptive health reasons. That inadequate illumination results in damage to the eyes is rarely stated directly, although the literature cited above is rife with threats of "permanent injury,"[1] "tragic toll"[36] and "deeply hidden penalties"[37] that "can only be revealed by careful and unique scientific research,"[38] and with such suggestive illustrations as a blind man being led by a Seeing-Eye dog.[39] The responsibility of the medical profession is here obvious, but it is difficult for the average physician, who has neither the time nor the interest to review the literature, to keep his equanimity in the face of the abundant propaganda. There is no generally acceptable evidence that poor illumination results in organic harm to the eyes any more than that indistinct sounds damage the ears or faint smells damage the nose. To be sure, prolonged concentrations under inadequate seeing conditions, estimated at less than 3 foot-candles,[35] will result in fatigue. But the fatigue is one of attention, and the complaint is generally a headache. There is no evidence that this results in cataract, glaucoma, separated retina or any of the other organic diseases of the eyes. For years it has been suggested that the so-called "unhygienic" condition of poor illumination and much close work result in nearsightedness. Luckiesh accepts this unqualifiedly, and in presenting it to the public, proposes another etiologic agent: reading from glossy paper.[19] There is no satisfactory evidence for the theory that nearsightedness is the result of much close work, poor illumination or reading print on glossy paper, whereas there is much evidence against it.[40] There is no proof that what is generally called "poor illumination" results in organic disease of the eyes.

It has been asserted that "the new science of lighting becomes a humanitarian movement which should engulf every human being,"[41] but for those who have not taken their lighting seriously and who in consequence do not have one of the light meters, which Luckiesh augurs will be part of every well-equipped home,[42] the following information may be of interest. A new 40-watt frosted bulb will provide an illumination of 10 foot-candles at 23 inches, 15 at 19 inches, and 25 at 14 inches.[43] Although this seems adequate for normal purposes, a somewhat more intense illumination may be desirable in certain conditions such as in uncorrected presbyopia, in which the smaller pupil, induced by the brighter light, makes up in part for the lack of visual acuity from insufficient accommodation.[44] This, of course, is not necessary when suitable glasses are worn. But the important thing is not that light requirements vary in different people, which in itself might be held as sufficient evidence to invalidate a universal mechanical meter,[45] but that when a person, as a presbyope, is benefited by more illumination he instinctively turns on more light. And he does this without any knowledge of what is happening to his heart rate, pupil and so forth. Contrary to the contentions of Luckiesh and Moss, there is no valid reason for believing that man cannot judge the amount of illumination that gives him easy and comfortable seeing. There seems little justification for the analogy that just as man needs a thermometer for measuring temperature or a pair of scales for weighing a beefsteak,[46] he needs a mechanical device to tell him how easily he reads. Anyone who has had the limited experience of getting used to a hot bath knows how much more variable the sense of warmth is than a thermometer indicates. Similarly, it is the person, with all the variables that make up a human being, who is going to do the reading, not the meter.[46]

Eyestrain

Most patients and some physicians believe eyestrain to be a well-defined entity, having specific causes, manifesting characteristic signs and symptoms, being readily detectable by objective examination, and leading to permanent damage of the eyes if untreated. To the ophthalmologist, on the other hand, eyestrain, although a convenient term, has less positive implications. "To the patient it has a definite and satisfying meaning, but to the doctor who employs it, it is almost if not quite meaningless, so that by its use he [the ophthalmologist] does not really commit himself in any way."[47] Here an attempt will be made to describe this ill-defined syndrome, to discuss some of its causes, and to suggest its physiologic background in the light of modern ophthalmologic thought. There is little doubt that the description to be given accords with the majority of ophthalmologic opinion, but the responsibility for the interpretation of the symptoms must, in view of the scarcity of literature on the subject, rest with me.

The chief symptoms and signs that make up

the syndrome of eyestrain are headache, sandy feeling and aching of the eyes, pulling sensation in and about the orbit, unusual sleepiness, and hyperemia of the globe and lids. None of these or of the many other symptoms that might be included in the syndrome are specific.

The alleged causes of eyestrain are legion, but refractive errors, muscle imbalances and faulty illumination have received most attention. Any visual task requiring concentration, especially when some extraneous influence makes this difficult, is potentially a source of eyestrain. It is not so much the refractive error or other handicap to vision that is responsible for the symptoms of eyestrain as it is the effort that is made to overcome the handicap. Whether or not glasses are indicated cannot be determined by knowledge of the refractive error and visual acuity alone. The effort that the patient exerts to overcome what handicap he may have must be taken into consideration. This is a very practical matter, and it is failure to take cognizance of it that has led to much unnecessary giving and changing of glasses, especially by nonprofessional refractionists. Persons with small refractive errors, especially low-grade hyperopia and astigmatism, have severe symptoms of eyestrain, whereas those with errors so large that presumably no voluntary effort is made to correct them have no eyestrain. Indeed, the latter may have eyestrain only after wearing glasses for the first time. By the same token, persons with normal ocular mechanisms may on occasion have typical and genuine strain on using their eyes. The symptoms of eyestrain, thought to be due to errors of refraction but also occasionally present with no such error, are thus attributable not so much to the peripheral organ as to a central mechanism that attempts to correct for the defect. It is possible that this central mechanism may be invoked and may produce typical symptoms of strain without any change in the eyes themselves, thus attesting to the purely central origin of the discomfort. This is seen, for example, when an out-of-focus picture is projected on the screen. Here there is no question of accommodation, muscular imbalances and so forth, which does not apply to an in-focus picture, but it is distinctly less comfortable to view.

Although the evidence for the central origin of eyestrain from refractive errors would probably be accepted by most experienced refractionists, the symptoms arising from muscle imbalances or from exertion of the extraocular muscles are generally thought to arise in the muscles themselves. Certainly the discomfort feels as though it came from the region of the orbit. Furthermore, fatigue of striated muscles elsewhere in the body is commonly believed to be due to accumulation of metabolic waste products and consequent stimulation of the nociceptors in the muscles. A number of observations, however, are apparently irreconcilable with this idea of the peripheral origin of discomfort in the eye muscles. A few may be cited. If a person maintains upward gaze for any length of time he feels a discomfort about the eyes that may culminate in a headache. But a person who, owing to a supranuclear lesion, cannot look up has the same symptoms merely from the continuous attempt to look upward. Here there is no muscle contraction or accumulation of metabolic waste products in the muscle tissue. Another striking example is afforded by nystagmus, in which condition the eyes are continually moved back and forth for hours without fatigue, whereas similar voluntary movements by a person with normal ocular motor mechanisms would be extremely fatiguing. There is thus a good argument for the hypothesis that eyestrain from muscular exertion is, like that from refractive errors, of central rather than peripheral origin.

The nature of the central process that I have called "visual effort" is far from clear, but it is known that such a process occurs and that it is not motivated solely by the eyes. The alpha rhythm from the occipital cortex, as detected with the electroencephalogram, may be abolished merely by the attempt to see in the dark just as it is in response to a peripheral visual stimulus.[48] An interesting case has also been recently reported in which a bruit over an occipital hemangioma was exaggerated by the attempt to see.[49] Both these observations are qualitative indications of some of the central processes taking place with visual effort. It is in such terms that eyestrain may eventually be explained.

What has been said of eyestrain from refractive errors and muscle disturbances probably applies to strain from other causes as well. It is an effort syndrome arising from the performance of a visual task, usually in the presence of some handicap, and it makes little difference whether the handicap is one of refractive error, glaring lights, distracting noises or difficult texts. Eyestrain is a central process related to concentration. The presence of some extraneous factor rendering the visual task difficult might be appropriately called mental glare, inasmuch as it consists in something other than the visual task imposing itself on consciousness. Instead of being a simple entity, eyestrain is thus a complex process extending well into the psychologic realm, and is certainly not amenable to evaluation merely by objective examination.

Granted the validity of this concept, it is apparent that although eyestrain may cause various psychologic disturbances,[50] it does not result in any organic disease of the eyes. Nor would one expect that organic ocular disease from whatever cause would be made worse by eyestrain any more than by other psychic annoyances. It is also apparent that the treatment of eyestrain is symptomatic only. Glasses are analogous to crutches, and important as their service is in this capacity, they have no further therapeutic value so far as the eyes are concerned. The common allegation that "wrong glasses" have ruined one's eyes is entirely without foundation. This concept of eyestrain also suggests the error in ascribing a medical implication to many of the maxims that have arisen from purely intuitive considerations. The reason that one should write with the light over one's left shoulder is that this arrangement makes the shadows least bothersome. The opposite holds for left-handed persons. As for reading, it makes no difference over which shoulder the light comes. Nor does any harm, other than perhaps discomfort, come from reading in bed. The same applies to reading on the train. Many of the misconceptions that have arisen as presumed health or sight precautions are clarified by an understanding of the psychologic nature of eyestrain.

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Much pseudoscientific evidence pertaining to illumination and eyestrain has been generally accepted and enshrouded with medical implications. Unfortunately, the medical profession has concerned itself little with the problems arising from these beliefs. There is, for instance, little or no discussion of them in ophthalmic or medical texts. It is hoped, therefore, that this review will bring to the attention of those who are the final arbiters of public health a reasonable and critical judgment in these matters.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27111

PRESENTATION OF CASE

First Admission. A sixty-one-year-old physician entered the hospital for study.

The patient was well until ten years previously, when he contracted lobar pneumonia; shortly afterward he developed a phlebitis in his right leg following a fall. During the next ten years he had six more attacks of phlebitis, four in the right leg and two in the left. At the end of this period he entered this hospital complaining of gas in his stomach occurring in the midafternoon and associated with nausea and belching, without vomiting. He had had these symptoms for two months and also complained of swelling and cramps in his legs, which he attributed to the old phlebitis. On examination at this time, the fundi showed slight nicking and tortuosity of the vessels, but the peripheral arteries were not palpable. The thyroid gland was slightly enlarged and appeared to contain several nodules. Examination of the heart was negative except for a soft systolic murmur at the apex; the blood pressure was 150 systolic, 84 diastolic. Examination of the urine and blood was negative. An electrocardiographic recording showed a normal rate and rhythm. The QRS_3 complex was of slightly low amplitude, with very slight slurring of QRS_2 and QRS_3. T_1, T_2 and T_3 were upright, and the chest lead was normal. X-ray studies of the sinuses, skull and gall bladder were negative; a gastrointestinal series showed multiple diverticula of the sigmoid.

Second Admission (three years later). The patient carried on for the next two and a half years, at the end of which he presented his physician with an electrocardiogram taken in California two months previously following an attack of pain said to be typical of coronary thrombosis. This recording was distinctly abnormal and was interpreted as being more in keeping with a basal infarct, including partial auriculoventricular block, than with an acute cor pulmonale from pulmonary embolism. His physician found moderate pitting edema of both legs from chronic varicosities. There was a very slight aortic systolic murmur, but the heart seemed normal in size, sounds, rate and rhythm; the blood pressure was normal. An electrocardiogram was now essentially normal, with slight inversion of Lead 3. Fluoroscopic examination showed a full-sized heart with normal aorta and clear lungs. Six months later, the patient returned to his physician merely for the purpose of a routine check-up; fluoroscopic examination showed a large shadow in the middle of the right lung. The temperature, heart, and electrocardiogram were normal. An x-ray film of the chest taken the next day showed the diaphragm to be normal in outline, position and excursion. The right lower and middle lobes and the base of the right upper lobe contained multiple sharply defined areas of consolidation. These areas were in contact with the pleural surfaces and moved normally with the lung. There were at least seven separate areas of density. The right lower and middle lobes were probably reduced in size, the lower more so than the middle. The septum between the lower and upper lobes was convex downward and posteriorly. The largest areas of consolidation were in the superior margin of the middle lobe and in the apex of the lower lobe. There was no evidence of fluid. The mediastinum was moderately displaced to the right. The apex of the heart was sharp and pointed, suggestive of a small, sharply defined area of consolidation in the adjacent lung. The left lung and upper portion of the right were otherwise clear. The aorta was normal. Three days after this examination, the patient had a flare-up of his phlebitis in the right leg and was admitted to the hospital.

The right saphenous and femoral veins were divided. An uneventful convalescence followed, but one month later the patient noticed that he was slightly dyspneic and complained of pain in the right mid-chest anteriorly. X-ray examination of the chest showed what the roentgenologist interpreted as fresh pulmonary infarcts. The right common iliac vein and the hypogastric vein and artery were sectioned. Following this procedure, the patient developed a rise in temperature, with signs of consolidation in the right middle chest, and Type 3 pneumococci were isolated from the sputum. He was given sulfapyridine with apparent improvement, but continued to run a swinging temperature up to 101°F., the pulse running between 90 and 100, the respirations around 30. Approximately one month after the operation a blood culture grew an unidentifiable pneumococcus. The additional and relevant physical findings at this time were as follows: The chest showed limitation of motion on the right side anteriorly and posteriorly, and there was dullness to percussion from the second to the sixth rib

on the right anteriorly. The breath sounds were slightly diminished throughout the right chest anteriorly. Posteriorly on the right there was dullness from the sixth spinous process to the ninth, where percussion elicited slight tenderness. Tactile fremitus was absent at the right base, and the breath sounds were diminished progressively from the sixth spinous process down, becoming absent below the ninth. Examination of the heart was unchanged. The extremities showed edema, with marked pigmentation over the anterior surface of the shins. Over the next ten days the chest signs improved so that at the time of discharge there was a definite increase in resonance both anteriorly and posteriorly on the right, with less suppression of breath sounds over the involved areas. A positive blood culture was not obtained again.

Third Admission (four months later). The patient returned to the hospital for a check-up and because of pain in the right lower leg and foot of one month's duration. The pain had started in the big toe, and gradually extended to include the lower leg. It was worse at night and during rest, and was relieved by walking. The patient was a little dyspneic on exertion. Examination of the chest showed limited expansion on the right, with dullness, diminished breath sounds and coarse wheezes on this side. The right toes and foot became red when in the dependent position and blanched promptly on elevation. There were varicosities around the ankle, with slight edema, and tenderness was present over the dorsum of the right foot and lower leg, with limitation in dorsiflexion at the ankle. Pulsation was good, and there were no obvious temperature differences. Examination of the urine and blood was negative; the sedimentation rate was normal. The nonprotein nitrogen of the blood serum was 22 mg. and the uric acid 5.1 mg. per 100 cc. An electrocardiogram was within normal limits. X-ray study of the chest showed that the large area of density in the right middle lung field had now completely disappeared, leaving only a few linear bands of density extending laterally and downward from the hilus. The diaphragm was irregular in outline and limited in excursion. The left lung was clear, and the heart shadow slightly displaced to the right. The heart showed slight prominence in the region of the left ventricle. There was no evidence of fluid or active disease. X-ray films of the legs were essentially negative. The patient was discharged three days later with a diagnosis of peripheral neuritis.

Fourth Admission (thirteen months later). Eight months before this admission, the patient had developed a cold that went to his trachea, with the result that he had difficulty with wheezing respirations, and from time to time a little "asthmatic breathing." An x-ray film taken five months before entry showed a high right diaphragm, with numerous thin lines at the site of the large areas of consolidation described on previous examinations. There were no remaining areas of consolidation and no new lesions. Three weeks before admission the patient became short of breath on exertion, and, in addition, had attacks of nocturnal dyspnea during which he was compelled to sit upright, although, in general, lying flat gave no trouble. This dyspnea was accompanied by a tiring, brassy cough productive of a little white frothy sputum and a heavy feeling in the chest under the sternum. The onset of these symptoms was not sudden, nor was there pain.

On examination, the patient was cyanotic, with some increase in respiratory rate, and a slight respiratory crow was audible. Examination of the heart was negative except for slight displacement to the right; the blood pressure was 130 systolic, 82 diastolic. The right chest was retracted, and posteriorly at the base the percussion note was dull and the breath sounds, vocal and tactile fremitus absent. The liver was palpable two fingerbreadths below the costal margin on deep inspiration. Examination of the urine and blood was negative; the red-cell count was 5,450,000, the sedimentation rate normal, and the vital capacity of the lungs 1741 cc. An electrocardiogram was within normal limits. X-ray examination of the chest showed the diaphragm to be obscured by a homogeneous density, which shifted when the position of the patient changed. There was consolidation at the apex of the right lower lobe and in the region of the anterior portion of the right middle lobe. The heart was to the right of the spine, without shift of the mediastinum. The right side of the diaphragm moved very little. The left lung was clear except for slightly diminished radiance at the base. X-ray study of the gastrointestinal tract showed the esophagus, stomach and duodenum to be essentially normal. The esophagus was deviated toward the left at the lower third. The right chest was tapped on two occasions, but only once was fluid obtained, and then but 3 cc. were withdrawn. This fluid contained 2250 red cells, 6600 lymphocytes, and 400 polymorphonuclears per cubic millimeter. No tumor cells were identified by the pathologist. The patient continued to crow and wheeze on respiration, the sounds being heard over the superior mediastinum and remaining audible in whatever position he assumed. Examination of the trachea and vocal cords was negative. The patient was discharged on the ninth hospital day.

Final Admission (three weeks later). In the interval, the dyspnea, asthmatic breathing and cough with slight sputum had persisted, and had indeed become slightly more pronounced. Two weeks before admission the patient began to suffer from profuse sweating and periods of chilliness without fever. A blood examination was said to have shown malarial parasites, and he was given a course of atabrin (15 tablets). This was followed by a diminution in size of the "right lobe of the liver," but did not change the clinical course. Eight days before admission, he developed in the lower chest a sudden pain, which was not associated with an increase in dyspnea. This pain lasted four days and was relieved promptly by manipulation of the vertebrae. However, at this time he complained of a sudden right epigastric pain associated with nausea and relieved by "Ludozan." Subsequently any solid food initiated this pain, which was accompanied by extreme localized tenderness. On one occasion, two days before admission, the patient raised a small amount of blood-streaked sputum. With the increase in dyspnea there was some orthopnea, and the pulse became intermittent at 90; the blood pressure was 110 systolic, 70 diastolic. The patient was given 0.5 cc. of digalen, which lessened the right-upper-quadrant abdominal tenderness and cleared the rales heard in the chest. The pulse became regular at 84.

On examination the patient showed striking evidence of weight loss. He was dyspneic, with a rasping, asthmatic wheezing precipitated by any movement. He coughed frequently, producing a thick mucopurulent sputum. There was slight cyanosis, but no obvious venous engorgement of the neck veins. The trachea was displaced to the right. The right chest was flat with limited expansion, and there was dullness to percussion from the third interspace down, with diminished breath sounds. Posteriorly on the right there was a dull to flat percussion note from the lower scapular region down, with absent breath sounds and tactile fremitus. Slight hyper-resonance was present at the right base. The heart was displaced to the right, and a rough systolic murmur was heard along the left sternal border. The pulmonary second sound was slightly greater than the aortic second, the rhythm was regular, the blood pressure 122 systolic, 65 diastolic. Examination of the abdomen revealed tenderness 5 cm. below the xiphoid and slightly to the right. The liver margin was palpable 4 cm. below the costal margin in the mid-clavicular line and was diffusely tender. Peripheral edema was absent; the right leg was somewhat larger than the left, but was not tender. No pulsation could be felt in the right dorsalis pedis artery, but it was present on the left.

The temperature was normal, the pulse 98, and the respirations 35.

Examination of the urine showed a + test for albumin. Examination of the blood showed a red-cell count of 4,410,000 with a hemoglobin of 15.1 gm. (photoelectric-cell technic), and a white-cell count of 9500. The nonprotein nitrogen of the blood serum was 19 mg., the serum protein 6.1 gm., the calcium 11.1 gm., the phosphorus 3.9 mg. per 100 cc., and the chlorides were 97.8 milliequiv. per liter. The sedimentation rate was normal. Examination of the stools showed a + guaiac test.

X-ray films of the chest showed very little, if any, change in appearance since the last observation. There was definite evidence of fluid and an extensive destructive process in the right lung. The left lung appeared normal.

On x-ray study of the abdomen, the outline of the diaphragm was not visible on the right side, and there was dullness extending a little below the twelfth rib. The kidney on this side was easily seen, appeared normal in size and shape and was apparently displaced downward so that its lower pole overlay the crest of the ilium. The shadow above it was of homogeneous density and was possibly due to an enlarged liver. There was no abnormal shadow on the left side. There was a moderate amount of gas in the colon and small bowel. The spine showed hypertrophic changes.

An electrocardiographic recording showed a normal rhythm with a rate of 100. T_1 was low and slightly diphasic. S-T_2 was sagging with slight late inversion. T_3 was inverted, R_4 upright and T_4 inverted (for the most part, late).

The patient never improved, continued to complain of severe attacks of epigastric pain and was weak, slightly cyanotic and dyspneic, with a troublesome cough. Four days after admission he again raised rusty sputum and continued to do so thereafter; the white-cell count was 15,800. He was placed in an oxygen tent, given diuretics and heparin. On the thirteenth hospital day the patient passed a tarry stool, and for ten days thereafter the stools were guaiac positive. The temperature was now 101°F. At this time the right elbow became painful on any motion and the left forearm and hand slightly swollen; edema of the feet developed, more marked on the left, with tenderness in the inner right mid-calf. Latterly the patient became disorientated, peripheral edema increased, the liver grew larger and tenderer, and the temperature rose to 102°F. Death occurred on the thirty-seventh hospital day.

Differential Diagnosis

Dr. William B. Breed: Ten years before the final admission this patient came in complaining of gas in the stomach with nausea and belching, but no vomiting. The x-ray films showed merely diverticula of the colon. The electrocardiogram showed nothing specific, merely slurring of the QRS_2 and QRS_3. The patient was then apparently discharged from the hospital with no diagnosis. Certainly no one found out why he was having indigestion.

Three years later, at the age of sixty-four, he returned to the hospital with the story that he had had pain in the chest, and produced electrocardiograms that had been interpreted by somebody as representing basal infarction with partial auriculoventricular block. The heart at this admission, however, was entirely normal, and another electrocardiogram showed only slight inversion of T_3, which means nothing. I should like to know who interpreted the other electrocardiogram and whether or not we can safely say that the patient had had a basal infarction of his heart. I am inclined to doubt it. He had a flare-up of phlebitis during this admission and was operated on, the right saphenous and femoral veins being sectioned. Obvious pulmonary infarction was found by x-ray examination. Then the common left iliac vein and artery were sectioned. This was followed by signs of consolidation in the right chest, the sputum containing Type 3 pneumococci. At that time an unidentified pneumococcus was recovered from the blood; then the chest cleared, I assume, and the patient went home.

About four months later he had pain in the right lower leg and foot for one month; it was worse when resting and relieved while walking. There were varicosities about the ankle. An electrocardiogram was reported at that time as normal, and the blood chemical findings were perfectly good. The patient had no renal difficulty. The x-ray studies at that admission should be of interest. He was discharged then with the diagnosis of peripheral neuritis, which I am inclined to doubt. Thirteen months later, he returned to the hospital with the story that eight months previously he had had a cold, which had gone to his trachea, and that he had been having asthmatic breathing on and off ever since that time. Additional x-ray films showed numerous lines at the site of the mass, which had disappeared. Three weeks previously, the patient had had attacks of nocturnal dyspnea associated with brassy cough and a heavy feeling under the sternum, but no pain. At this time he was cyanotic, and the heart was displaced to the right.

At the fourth admission, the chest was tapped, and 3 cm. of fluid was withdrawn, which showed 2250 red cells and 6600 leukocytes with 400 polymorphonuclears, and which certainly was not just blood and must indicate either inflammation or some malignant process. No tumor cells were identified by the pathologist. I think it would be of value to try to get some order out of this chaos by taking up the various systems, discussing first the one that is least likely to cause this trouble. That, I believe, is the metabolism, as reflected in the chemical examinations. The phosphorus is all right, the nonprotein nitrogen is normal; the calcium is 11.1 mg. per 100 cc., which is very slightly above the high normal of 10.8. With that single finding I cannot go any farther, but I do not see that it is of any significance.

I think the next group of symptoms or signs I should like to take up would be those pertaining to the cardiovascular system. It seems to me very unlikely that this man had had a basal infarction and then had a return of the electrocardiogram and other findings to normal. My experience is that younger people with infarction have a return to a normal electrocardiogram more frequently than people over sixty. It is not often that the signs of infarction disappear in a man of this age. Therefore, I am doubtful about this coronary occlusion. The cardiac situation does not seem to me to be very prominent. The patient had a fall in blood pressure, and toward the end probably did have congestive failure, but I do not believe that he suffered any accident to his coronary arteries.

The pulmonary situation in the first part of the illness is quite simple. The patient had phlebitis and recurring pulmonary infarction, which we know about; and up to the latter part of the illness the pulmonary situation seems quite clear. The x-ray films showed definite clearing of the initial lesions. Then latterly he developed again a definite pathologic process in the right chest, which caused pulling of the heart to the right, indicating a collapse of the middle lobe and perhaps the right lower lobe. I believe that this was some destructive process in the right lung and was the main cause of his death.

I should like to have Dr. Hampton interpret the x-ray films.

Dr. Aubrey O. Hampton: I looked over these films carefully. The patient had many examinations and many films. These are the most interesting. If you want to know anything about the others, I have them here. At the first examination there were multiple, sharply defined areas of consolidation in the right middle and lower

lobes. They corresponded to the margins of these lobes and had the general shape and appearance of infarcts. There was no pleural reaction, however. Here on the left side is a short linear shadow continuous with a v-shaped configuration of the apex of the heart, which we thought was a healed infarct and which, perhaps, was related to the cardiac episode. The patient might have had precordial pain and cardiac embarrassment with it. One month later this picture in the chest had not changed. Another film taken twenty-three days later, following the first operation, showed obliteration of the previously clear costophrenic angles. If one compares the lateral views, the shadow of the heart is covered by new sharply defined masses, so that we may assume that there were two more infarcts following the first operation. At that time the patient said, "I have finally had the pain you have been trying to get me to admit." Nearly two and a half months later, following the second operation, this film — one of the many films at this time — showed pleural effusion and reduction in size of the masses in the right lung. Four months after that all the lesions in the lung had practically disappeared, leaving numerous lines similar to the one noted at the left border of the heart, so that we then assumed that the infarcts had healed.

DR. BREED: So far we are even.

DR. HAMPTON: These are the films of the last admission. There is now a change in the whole picture. The patient came in with about the same evidence of pleural disease that he had following the second operation. There are also masses in the right mid-lung field, and in addition a circular area of diminished density, which looks like a cavity. The trachea, heart and mediastinum have all shifted a little farther to the right. We examined the esophagus and trachea, trying to discover the cause of the discomfort and wheezing, but could find very little wrong, only a little esophageal displacement where it crossed the aorta. The stomach was normal, and the shadow of what we thought was his liver was large. The kidney was displaced downward. The spleen was not enlarged so far as we could tell. This final film was taken one month later, and shows numerous, irregular cavities in the region of the middle and lower lobes. Of course the heart would shift to the right if the lung were destroyed.

DR. BREED: I think it is reasonably clear that this patient had a serious chronic condition in the right lung. Whether it was due to primary cancer in the bronchus or to a primary destructive lesion farther down, or whether it was metastatic cancer or multiple abscesses in the collapsed lung, I am sure I have not yet a clear idea, but I am reasonably certain that this man died of pulmonary disease primarily. We then come to the question of tarry stools, and this very peculiar complaint of tenderness and pain in the same spot when he swallowed food. Is that a good description of what he really showed? You will admit that it is a peculiar symptom.

DR. TRACY B. MALLORY: Can you answer that, Dr. Stetson?

DR. RICHARD P. STETSON: After eating, the patient had spontaneous pain, which he localized in the right epigastrium, and there was a spot of persistent tenderness just to the right of the midline.

DR. BREED: Let us consider the gastrointestinal tract. When he first came in, the complaint was indigestion, and nothing more is said about the tract throughout the whole long illness up to the point of this situation toward the end. It seems a far cry but it is possible, I suppose, that he had some cancer in his gall bladder, with metastases to the lung, and possibly later erosion of the esophagus or some other portion of the gastrointestinal tract, with the production of tarry stools. I cannot believe that he died suddenly from hemorrhage, although he did have this large tarry stool. Though the intestinal tract may have been secondarily involved, I do not believe it was a primary feature of the disease. I think that the patient died of cancer primarily in his chest, which may have been complicated by multiple abscesses behind an obstructed bronchus. Although he probably had some terminal heart failure, I doubt if this man had an occlusion of an artery in his heart.

DR. MALLORY: I should be glad to have you cross-examine any of the expert witnesses. They are nearly all present — Dr. White, Dr. Faxon, and Dr. Stetson.

DR. BREED: Dr. White, you are first. You were satisfied that this patient had had a basal infarct?

DR. PAUL D. WHITE: I was the first of the chain, and then was less in touch with him as the heart began to fade from the picture. I saw him first in May, 1938. He had come back from California with the story and electrocardiogram typical of something happening suddenly in the chest. The electrocardiogram showed auriculoventricular block and deformity of Q and T (prominent Q's and inverted T's) in the second and third leads. The auriculoventricular block seemed to indicate not an acute cor pulmonale but basal myocardial infarction in all probability. We had never encountered heart block before in acute dilatation secondary to pulmonary embolism. My first diag-

nosis was therefore coronary thrombosis in May, 1938, and phlebitis of twelve years' duration. I checked up six months later, when he felt perfectly well, but I was amazed at that time to find a large shadow in the right lung. I sent him then to Dr. Hampton. You have heard the rest of the story from him.

DR. BREED: Were you surprised to see the heart and electrocardiogram return to normal?

DR. WHITE: No, that happens occasionally with basal myocardial infarction, so that despite the fact that the electrocardiogram was normal in May and again in December, I did not change my original diagnosis but added that of pulmonary infarction.

DR. DONALD S. KING: Could it all be the result of septic infarction in that area?

DR. BREED: With multiple abscesses? I suppose it could. I do not know how one can explain the tarry stool and positive guaiac tests on that basis.

To go back to the heart: The last electrocardiogram was perfectly consistent with digitalis effect or with, perhaps, some coronary disease, but I cannot interpret it as coronary occlusion. Of course, the patient may have had one after the last electrocardiogram, but there is nothing in the history to indicate it.

DR. WHITE: I think your reasoning is excellent.

DR. BREED: It may be excellent, but I do not think it is going to produce anything brilliant here. I have committed myself to cancer, either primary or secondary in the right chest. I should be inclined to think it was primary and not secondary.

DR. GEORGE W. HOLMES: One of the things that has bothered Dr. Breed is this intestinal hemorrhage. I should like to suggest the possibility of embolus to the mesenteric arteries.

DR. BREED: I did consider it, but there was nothing in the story to indicate its presence. The patient lived for ten days after the positive guaiac, and nothing was said about an acute abdominal situation. I hate to surrender the explanation of this tarry stool but I have not a thought, except erosion of the gastrointestinal tract by cancer.

DR. KING: If you saw the last x-ray films and did not know the story, what diagnosis should you make, Dr. Hampton?

DR. HAMPTON: I should have to say primary carcinoma of the lung with metastases to the liver.

DR. KING: If you saw the x-ray films alone, you could not escape that diagnosis, but I do not know whether it is a good one.

DR. BREED: I am willing to make that diagnosis, but I am not going to say metastasis to the liver.

DR. HAMPTON: How are you going to account for the large shadow in the right upper abdomen?

DR. BREED: By his congestive failure.

DR. HAMPTON: His spleen is not enlarged.

DR. BREED: Well, what of it?

DR. HAMPTON: That is a very big liver, if it is the liver. I am not sure that it is.

DR. BREED: If you do not know, I should not be expected to, and these men will not tell me; I therefore do not think they knew either.

CLINICAL DIAGNOSES

Pulmonary infarction.
Bronchopneumonia.

DR. BREED'S DIAGNOSES

Cancer of the lung, ? type.
Erosion of gastrointestinal tract, ? site.
Phlebitis.
Pulmonary infarction (old).

ANATOMIC DIAGNOSES

Reticulum-cell sarcoma involving right lung, pleura, stomach, retroperitoneal tissues, mediastinum and trachea.
Thrombosis of inferior vena cava, common iliac, femoral and deep veins of legs, bilateral.
Arteriosclerosis, slight, aortic and coronary.
Cholelithiasis and choledocholithiasis.
Stricture of left ureter.
Hydronephrosis.

PATHOLOGICAL DISCUSSION

DR. MALLORY: To start with the negative findings: The heart was small, the coronary arteries capacious, and there was no significant amount of coronary sclerosis. On the positive side, this man was riddled with tumor. The largest amounts of tumor were in the right thoracic cavity, partly in the form of massive pleural involvement but also as a complete neoplastic replacement of the right lower and middle lobes. In the midst of the tumor were many small cystic cavities where the tumor had broken down. The liver was normal in size, small, — weighing 1500 gm., — and the mass that had been felt and interpreted on x-ray examination as liver was an immense mass of tumor in the gastrocolic ligament, which ran backward and was continuous with more tumor, chiefly centering in the nodes around the head of the pancreas and also destroying the pancreas itself. A peculiar finding was that all the mesenteric tabs in the sigmoid were infiltrated with

neoplasm. A significant amount in the posterior mediastinum had invaded through the trachea at one point, and a small tumor mass was present within the trachea. I therefore think that the wheezing and the asthmatic breathing were due both to intrinsic and to extrinsic narrowing of the air passages.

The probable source of the tarry stool was the stomach, which presented a most extraordinary appearance of about 50 separate small tumors in the mucosa, most of them with shallow ulcerations. The widespread distribution of the tumor masses and the lack of obvious primary focus suggested some form of lymphoma, and on microscopic examination we finally classified this as a reticulum-cell sarcoma. We were able to find in the left lung one linear scar, which Dr. Hampton pointed out on first entry and which I think represented a healed infarct. All evidence of the multiple infarcts that Dr. Hampton had described in the right lower lobe was entirely obliterated by the neoplastic growth. Despite the various venous ligations that this man had had, he still had a source for pulmonary emboli, since the thrombotic process extended well up into the vena cava. Incidental findings, which probably played no role in his symptomatology, were a stone in the common duct and a stricture of the left ureter, with a secondary hydronephrosis.

Dr. Henry H. Faxon: The operation was done at another hospital, and was attended, I am told, with a certain amount of difficulty. I believe that bleeding was encountered and prompted the surgeon to ligate the artery as well as the vein. The only other reason that could be advanced for sectioning the artery would be its alleged beneficial vasomotor effect on the deep phlebitis. Dr. John Homans, I believe, considers that in such cases section of an artery will be helpful, but I should be opposed to carrying out this procedure. I believe that lumbar sympathectomy or injection would be more satisfactory if the purpose were to secure distal vasodilation.

Dr. White: May I add one word about the electrocardiogram? During the last few years, I have occasionally found it difficult to tell the difference by electrocardiography between basal myocardial infarction and acute dilatation of the heart secondary to pulmonary embolism. As it turned out, there must have been that confusion here even to the point of believing that this patient had transient heart block with pulmonary embolism. Why that happened, I do not know, unless asphyxia could have been responsible, as has been proved in animal experiments. The terminal electrocardiographic changes could be explained by the serious final illness.

CASE 27112

Presentation of Case

A twenty-year-old girl entered the hospital complaining of pain and swelling in the right ankle.

The patient stated that seven months before admission, while she was walking, a sudden pain developed in her right ankle and was severe enough to initiate slight nausea. This was followed by swelling, fever and an area of redness on the outer surface of the ankle. These symptoms lasted for two weeks, then subsided, but three weeks later a similar episode occurred, apparently quite spontaneously. During the months before admission, the patient suffered six such attacks, and each time the symptoms gradually subsided, except for the persistence of slight swelling. Between times there was no pain, no inconvenience, but occasionally a slight twinge occurred in the ankle when she stepped in a certain way. Four days before admission, she happened to brush her ankle against a chair and there was pain, followed in several hours by swelling and tenderness. The pain increased, disturbing sleep, although somewhat relieved by elevating the part. She had not lost weight.

On examination the patient was well developed and well nourished and in no apparent distress. Examination of the heart, lungs and abdomen was negative; the blood pressure was 120 systolic, 80 diastolic. Over the right external malleolus there was a localized area of nonfluctuant swelling and redness, with a slight increase in surface temperature. Tenderness was present maximally at the lower end of the malleolus below the point where swelling was most marked. Active and passive movements of the joint were not painful or limited. No other joints were involved, and the regional lymph nodes were not swollen or tender.

The temperature, pulse and respirations were normal.

Examination of the urine showed a + test for albumin. Examination of the blood showed a red-cell count of 4,520,000 with a hemoglobin of 75 per cent, and a white-cell count of 12,100 with a normal differential. The sedimentation rate was normal. The serum calcium was 10.9 mg., the phosphorus 3.5 mg. and the protein 6.7 gm. per 100 cc., with the phosphatase 2.9 units. A blood Hinton test was negative, and agglutination reactions for *Eberthella typhosa, Salmonella paratyphi, S. schottmülleri* and *Brucella abortus* were negative.

Tuberculin tests were negative in dilutions of 1:10,000, 1:1000 and 1:100.

X-ray films of the right ankle showed that the distal 2.5 cm. of the right fibula was deformed into a multilocular cavity with coarse irregular trabeculation. The external malleolus was not involved, but the cavity probably reached the joint surface medially. The borders of the cavity were rather ill defined, and there was a fracture through its posterolateral wall, with periosteal new-bone formation in this area. A fairly marked diffuse soft-tissue swelling was present on the outer aspect of the leg.

X-ray studies of the chest, skull, bones of the forearms, wrists, humeri, lumbar spine and pelvis were negative.

On the seventh hospital day a biopsy was performed.

Differential Diagnosis

Dr. Richard H. Wallace: We have an apparently otherwise healthy young girl with a lesion in the lower end of the fibula, which has produced symptoms for seven months, and I believe that the first question we must try to decide is whether or not this can be an inflammatory lesion. There is nothing in the history to suggest a previous infection. The temperature, pulse and respirations are reported as normal. The patient had a slightly elevated white-cell count with a normal differential. A story of this duration with intermittent symptoms, chiefly pain, not only with activity but also with rest, is fairly unusual for infection. In addition, agglutination tests for one thing and another were negative. Although I believe it is possible, I think it is unlikely that she had infection. One other point in regard to infection: the initial onset of trouble was pain while walking. In view of the subsequent demonstration of a fracture line in the x-ray films, it seems possible that the initial symptom might have been the result of a pathologic fracture, which would be extremely unusual for the first symptom of osteomyelitis. Just in passing, I might say that it is extremely unlikely that this is either syphilis or tuberculosis.

We always have to consider parathyroid disease in any bone lesion. The calcium, phosphorus and phosphatase do not suggest parathyroid disease. It is certainly not simple cyst from the x-ray appearance, and the position in the bone is unusual, because simple cysts occur more toward the center of the shaft.

Benign tumor might be considered next. Giant-cell tumor can be ruled out because of the x-ray appearance and the fact that giant-cell tumor is usually confined to the epiphysis. This might conceivably be a chondroma, or, more likely, even an angioma of some sort.

This motheaten appearance suggests multiple myeloma, which with this duration should give involvement of bone somewhere else, and, again, the patient after this length of time probably would not be healthy in appearance. It could conceivably be lymphoma or something in the lymphoma series; reticulum-cell sarcoma is the likeliest possibility. Metastatic disease would have to be considered. It is not the usual appearance of the bone destruction that we see in metastatic disease. There is some new-bone formation here. Whether that is due to the old fracture or not is difficult to say. Osteogenic sarcoma is certainly a possibility. There is no great elevation of the periosteum, as we usually see in osteogenic sarcoma, and no ray formation, but osteogenic sarcoma frequently occurs without that.

Perhaps Dr. Holmes will help us at this point.

Dr. George W. Holmes: I think I know the diagnosis in this case. I have seen it before. Of course, in the lower end of the fibula there is an obvious lesion, which is almost wholly destructive and which occupies a considerable portion of the bone, but it does not look like an inflammatory lesion.

Then we come to tumor — whether it is benign or malignant. It is apparently a rather vascular tumor, and the only benign tumor that could possibly resemble it is an angioma, which, of course, is rare. The chances are in favor of primary malignant tumor.

Dr. Wallace: Should you say that there is a fracture through the lower end of the fibula, with new-bone formation at this site associated with it?

Dr. Holmes: It is not very obvious, but I think that there is one. If there is a fracture there, that makes it more difficult to interpret the significance of any periosteal proliferation.

Dr. Grantley W. Taylor: What are the characteristics that justify your saying that it is a highly vascular tumor? What are the x-ray characteristics?

Dr. Holmes: There is a good deal of bone absorption without bone proliferation. I have no very good grounds for my statement when you pin me down, but a vascular tumor shows a lot of bone destruction without much proliferation as a rule. There are trabeculae, moreover, throughout this tumor, whereas if it were a fibroma, or a fibrosarcoma one would expect destruction of all the bone. This apparently spreads irregularly

through the bone, leaving trabeculations, a thing that the vascular tumors are likely to do.

DR. WALLACE: Should you say that the periosteum is elevated to any great extent?

DR. HOLMES: There is some elevation.

DR. WALLACE: It comes down to the question of whether or not this is osteogenic sarcoma. The local signs are in keeping with that diagnosis—the redness and increased heat locally. The pic-

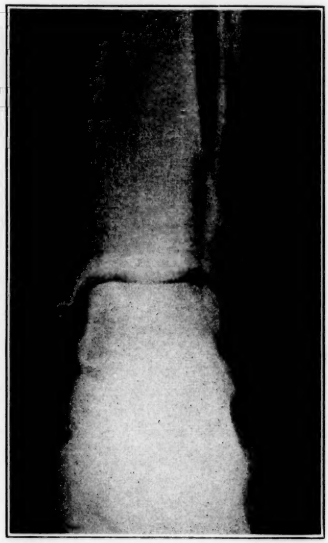

FIGURE 1.

ture is a little confused by the fracture. One ordinarily would expect a little more new-bone formation with osteogenic sarcoma of seven months' duration.

A likely possibility is, I believe, a Ewing's sarcoma, which can and occasionally does occur in this position in bone. The story of intermittent pain and local signs is common in Ewing's tumor. Early fracture is not unusual in weight-bearing bones. The slightly elevated white-cell count, with the normal differential, is in keeping with Ewing's tumor. I might say that, so far as bone tumors are concerned, elevation of the phosphatase is associated with new-bone formation. Of course, the reading is normal in this case, but in osteogenic sarcoma the readings vary from time to time, so that it is not necessarily elevated at any given reading. One other point about Ewing's tumors is that they are less likely to have new-bone formation. The onion-skin appearance in the region of the periosteum that we frequently see in Ewing's sarcoma is not present here. However, we have seen this appearance in osteogenic sarcoma and the ray formation commonly associated with osteogenic sarcoma in Ewing's tumor. I believe, with all the evidence at hand, that a final conclusion could be reached only by biopsy, but I shall say that I consider the likeliest diagnosis to be Ewing's tumor.

DR. CHANNING C. SIMMONS: Like Dr. Holmes, I do not know that I am qualified to discuss this case, for I saw it clinically. I made the correct diagnosis, not on the evidence submitted, but because I had seen a similar case some time before. In all cases of bone disease, one has to consider four general possibilities: generalized bone disease, metastatic tumor, an inflammatory condition and primary bone tumor, either benign or malignant. In this case, with the data we have, metastatic tumor and generalized bone disease may be ruled out, which brings us down to an inflammatory condition or tumor. The patient had pain and evidence of tumor in the area for some time, with redness. I do not know whether or not she ever had a temperature, but she had none on admission to the hospital. On examination, however, she had a slightly elevated white-cell count, which might be due either to inflammation or to bone tumor, for a malignant tumor occasionally gives an elevated temperature and white-cell count. From the story she obviously had had trouble with the ankle for some time, followed by an acute attack of pain probably owing to the crack and tear through the periosteum. Of the inflammatory conditions we have to consider syphilis, tuberculosis and osteomyelitis. The blood Hinton test was negative. I do not think it is tuberculosis, for the x-ray film does not look like it. Osteomyelitis is a definite possibility. The benign bone tumors, with very few exceptions, cause no pain. The exception is giant-cell tumor and sometimes a central osteoma. I have never seen a central osteoma, but they are said to be painful. This patient had a bone-destructive lesion, demonstrable by x-ray, and a normal temperature. I believe that it undoubtedly is a malignant tumor of some form, either osteogenic sarcoma or Ewing's sarcoma. In giant-cell tumor with bone destruction, the destruction is usually very sharply marked at the top, with a sharp angle at the periosteum, and extends to the joint cartilage. Ewing's sarcoma is rare in this situation. It usually is in the shaft, but can

occur in any situation. I believe that this is probably a Ewing's sarcoma, but think it is impossible to make a diagnosis without a biopsy.

Clinical Diagnosis

Benign giant-cell tumor of fibula.

Dr. Wallace's Diagnosis

Ewing's tumor.

Anatomic Diagnosis

Ewing's tumor.

Pathological Discussion

Dr. Tracy B. Mallory: A biopsy was performed on this patient, and a diagnosis of Ewing's tumor made. There was then considerable discussion about the best mode of treatment. Dr. Taylor can tell us how the argument ran in the tumor clinic.

Dr. Taylor: The discussion revolved about whether we were ever going to cure Ewing's tumor of the long bones by any means, and there was a great deal of pessimism expressed. Dr. Simmons has records in the registry of bone sarcomas of some cures by means of radical surgery. The response of the immediate local process to x-ray therapy is very gratifying in controlling the local manifestations of the disease until the patient succumbs to the pulmonary metastases. Control of the local process is all that can be secured by radical amputation, and the only hope in surgery is that one may obtain complete early removal of the disease and get rid of it before the pulmonary metastases have been established. It is perhaps reasonable to hope that effective local radiation might bring about the same result, namely eradication of the local process before dissemination has taken place. Whatever method of treatment is followed, the results have been uniformly very poor. I think that generally the radiologists believed that they would like to try radiation therapy on this case, and the surgeons thought that they would like to carry out amputation. A very poor third method of treatment was brought up only to be condemned, and that was the question of whether a resection of the involved bone could be carried out. I believe that the patient was finally treated by radiation.

Dr. Mallory: She is undergoing x-ray treatment now.

Dr. Simmons: In the registry there are records of some patients who lived without disease over five years. Two of these cases that I know of were controlled with radiation treatment. The others were treated by amputation. Unfortunately, statistics based on the cases reported to the registry are misleading, because surgeons are likelier to report favorable than unfavorable cases. I personally looked up 35 consecutive cases of Ewing's sarcoma treated by radiation or surgery — in all of which the patients are now dead.

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PHYSICIAN VERSUS PHARMACIST

Our esteemed, if relatively youthful, contemporary, the *Journal of the American Medical Association,* has taken a recent fling — and a deserved one, we believe — at our present most popular panaceas, the total vitamins.* The vitamins have had an interestingly prolonged build-up. Their existence was recognized, even if their nature was unknown, when Captain Cook first sailed the south Pacific and when the Dutch fishermen of the sixteenth century fed cod-liver oil to their babies to prevent that same rickets that Glisson so carefully described in 1650. It was not until 1911 that Funk first used the word, spelling it with a final "e," but these were not vitamins plus.

Cod-liver oil in those better days before horsepower replaced the horse cured only rickets; the juice of limes and other citrus fruits was used merely for its direct attack on scurvy; vitamin B, majestic, undivided and alone, made dietary war on the scourge of beriberi, until the alphabet gave substance to the conquest of pellagra by conferring a new letter on its conqueror.

The picture has been changing with increasing rapidity. Ultraviolet radiation has long since taken its stand as a practical counterpart of vitamin D; the vitamins no longer simply cure deficiency diseases but are essential contributors to positive health; vitamins E and K have been added to the list, and vitamin B has undergone lightning-like and repeated mitosis. The vitamin B complex, indeed, must now strut its part weighed in gammas, measured in international and Sherman-Bourquin units, and subdivided into numbered fractions — riboflavin and nicotinic acid, the filtrate factor, pantothenic acid, factor W, the factor of rat antidermatitis and the antigraying-hair factor.

A decade ago the vitamins constituted the coming therapeutic fact. Today, with a few endocrine hormones and the infant sulfa sisters sharing the stage, they are therapy itself.

The *Journal of the American Medical Association* tells us, in this same exposé of "vitamins plus," that more than two hundred vitamin products were pictured recently in an advertisement by a Chicago *department store.* No advertisement that we have yet seen informs us that all the necessary vitamins, in suitable quantities for those who are able to assimilate them, may be present in a well-balanced, properly prepared diet.

The vitamin preparations that can be sold over the counter and taken with presumable safety continue to capture the popular imagination, but it is also more than a half-truth that we who are medical practitioners have largely, and perhaps perforce, given over our therapy to the purveyors of proprietary preparations. Our investigative col-

*Editorial. Nonplusing "vitamins plus." *J. A. M. A.* 115:1639, 1940.

leagues, as we know, have been devising new methods in their laboratories and their clinics, but it is on the pharmaceutical houses and their detail men that we rely for the real ammunition of our practice, and to know where to buy and how to use the pills and potions, the panaceas and palliatives that scientific ingenuity and economic enterprise are supplying to us in such an endless stream.

We cannot master it all; we have little basis for selection; we know that it may take years to establish the real value of many of our vitamin preparations, our vaccines, our hormones and our drugs, and yet we cannot risk dropping out of the procession that so assuredly brands itself as medical progress.

Pharmacy is now far too complex for the corner pharmacist to supply the needs, much less the demands of the times, and the manufacturing pharmaceutical firm of good repute has become a very necessary adjunct to the effective service that we should all like to give. Is it, however, a part of this service that a single firm should list no fewer than twenty-four vitamin products running the gamut of the alphabet in various combinations from A to K, from alpha to omega and beyond?

POISON-IVY DERMATITIS

Dermatitis from poison ivy — the shiny, three-leaved pest draped abundantly over New England scenery from spring to winter — is always with us. The vine was probably a hazard to the comfort of the Pilgrims and, like sunburn, hay fever and mosquito bites, flourishes as a disturber of the peace of both natives and summer visitors. The variations in the susceptibility of a person to the plant and the latent period between the exposure to its poison and the involvement of the skin are emphasized anew in this issue of the *Journal*.

The monograph, *Dermatitis Venenata*, published in 1887 by James Clarke White,[1] the first professor of dermatology at Harvard Medical School, — and in this country for that matter, — presented the results of keen observation, an excellent botanical knowledge and a wide clinical experience with the effects of this plant on the skin. McNair[2] developed much of importance regarding susceptibility to the plant and the resulting dermatitis. Therapeutic remedies have been legion, and many suggestions for prevention have been made. Some success in prevention has been reported following the oral administration of a diluted tincture of the plant, in accordance with the suggestion of Schamberg,[3] and experiments are being made as to the efficacy of an extract[4] for oral administration. In some persons, immunity has been produced by the injection of an extract of the plant each year before possible exposure.[5]

More recently, Schwartz, Warren and Goldman[6] have recommended a preventive cream. Sodium perborate was added to a vanishing-cream base, and the mixture applied to the skin before exposure to the leaf. The volunteers on whom the fresh leaf was rubbed after the cream was applied did not develop any irritation. Only a few persons have thus far been tested, but the method deserves further trial by those who may be exposed to the plant. So far as is known, the effect of the paste on exposed persons has not been determined. This work resulted from an attempt to find a substance that would decompose or split up the complex chemical, urshiol, which is the active principal of the plant. Potassium permanganate will render the toxic agent inert, but discolors the skin; hence a search was made for a colorless preparation. This new method is relatively simple but must be used before every period of possible exposure. Although this is a handicap, any method that will prevent the dermatitis can well earn the praises of the many sufferers who are sensitive to this plant.

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MEDICAL EPONYM

Gruber–Widal Test

The Gruber–Widal test is based on the theoretical investigations of Max von Gruber (1853–1927) of the Department of Hygiene in the University of Munich, and their practical application by Fernand Widal (1862–1929). Von Gruber, in an article entitled "Theorie der activen und passiven Immunität gegen Cholera, Typhus und verwandte Krankheitsprocesse [Theory of Active and Passive Immunity to Cholera, Typhoid Fever and Related Diseases]," which appeared in the *Münchener medizinische Wochenschrift* (**43:** 206, 1896), concludes:

> By means of intraperitoneal inoculation with the killed culture of the vibrio of cholera, . . . the typhoid bacillus, and the colon bacillus, a high-grade and persistent immunity may be achieved in the guinea pig. . . . Antibodies are present in the blood and body fluids of the immunized animals. . . . The essential effect of the antibodies of the body fluids in these immunized animals consists in a destructive alteration of the enveloping structures of the bodies of the bacteria. This is shown by the fact that bacteria treated with these fluids become viscid, collect in large clumps and lose their individual motility.

Widal, in his article "Sero-diagnostic de la fièvre typhoïde [The Serum Diagnosis of Typhoid Fever]," which appeared in the *Bulletins et mémoires de la Société medicale des hôpitaux de Paris* (**13:** 561–566, 1896), after acknowledging his debt to Pfeiffer, Koll and von Gruber, says:

> We present a new method, which makes possible the diagnosis of typhoid fever simply by observing the effect of the patient's serum on a bouillon culture of Eberth's bacillus. . . . A small amount of blood . . . is collected under aseptic precautions; . . . the serum is decanted, and several drops are added to a tube of broth in the proportion of one part of serum to ten or fifteen parts of bouillon. . . . After twenty-four hours, the bouillon is only faintly clouded; a few floccules have settled to the bottom, and a whitish, rather heavy suspension is visible throughout the length of the tube. . . . Inspection of the tube is not enough; microscopic examination is also necessary. . . . The bacilli, instead of wriggling all over the preparation . . . are seen to be grouped, agglutinated together. . . . The diagnosis may also be made more quickly. . . . It may be so apparent that six drops of serum mixed with 4 cc. of culture, after being kept at room temperature for two or three hours, will produce the characteristic clumping to the naked eye. . . . The diagnosis must never be made until after microscopic examination. . . . If one drop of serum is added to ten drops of a culture of Eberth's bacillus, one may almost immediately demonstrate the characteristic bacterial agglomeration under the microscope.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SPECIAL MEETING OF THE COUNCIL

A special meeting of the Council of the Massachusetts Medical Society will be held in John Ware Hall, Boston Medical Library, 8 Fenway, Boston, on Wednesday, April 9, 1941, at 10:30 a. m., by vote of the Council on February 5, 1941, as provided in Chapter IV, Section 1, of the by-laws.

Business: To consider various changes in the by-laws recommended to the Council at its meeting on February 5, 1941 by the Committee on Medical Education and Medical Diplomas and by the Committee to consider new officers and by-laws.

Incidental business.

Robert N. Nye, *Secretary* pro tempore.

Councilors are asked to sign one of the two attendance books before the meeting. The Cotting luncheon will be served immediately after the meeting.

SECTION OF OBSTETRICS AND GYNECOLOGY*

Raymond S. Titus, M.D., *Secretary*
330 Dartmouth Street
Boston

Heart Disease in Pregnancy

Between 1 and 2 per cent of pregnant women in this country have organic heart disease. Many of these have minor valvular lesions of little consequence, and others have such severe cardiac damage as to involve considerable risk to the life of the mother and even more to the fetus. In addition to the relatively small group with clear-cut organic disease of the heart, there is a much larger group in whom the appearance of disease is simulated by functional systolic murmurs that may be quite loud and by symptoms such as dyspnea, palpitation and ankle edema that are not uncommon in normal pregnant women. It is the first task of the attending physician to determine, if possible, whether or not organic heart disease is present. A convenient rule of thumb is that organic heart disease exists if any of the following features are present: a diastolic murmur, definite cardiac enlargement, or auricular fibrillation. If these features are absent, the heart may be regarded as practically normal. There are exceptions to this rule, but they are rare.

Having satisfied himself that organic heart disease is present, the physician attempts to evaluate the degree of cardiac disability. The question

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

whether a given cardiac patient will be able to go through to term successfully can usually be answered better by the patient herself than by the intensity of the murmurs or other physical findings. If she gives a history of previous heart failure, she is likely to go into failure again before she reaches term. The older patients are, in general, poorer risks than the younger. Dr. Burton Hamilton has pointed out that heart failure occurs twice as often in women over thirty-five as in women under thirty-five years of age. Complications such as hypertension or pulmonary infection also add to the gravity of the prognosis. If the patient is young and her heart has never failed and there are no complications, the chances are good that she will carry her pregnancy through to a successful termination, regardless of the valvular lesion.

In considering the problem of the management of the heart patient in pregnancy, due weight should be given to recent work regarding the extent to which the gravid uterus burdens the circulation. Clinical and physiologic studies at the Boston Lying-in Hospital and elsewhere indicate that the work of the heart is increased about 40 to 50 per cent during pregnancy. Of great practical significance is the accumulated evidence that the peak of increased cardiac work is reached not at term but about one month before term. The subjective "lightening" observed by so many patients at this time appears to be associated with objective evidence of an easing of the load on the circulation, the cardiac output actually approaching normal before delivery.

In the light of this evidence, the practice in some clinics has changed from emptying the uterus as soon as a viable baby could be obtained to allowing the cardiac patient to go to term if possible. This policy has resulted in a decline in both maternal and fetal mortality.

The physical activity of all patients with organic heart disease should be rigidly restricted throughout pregnancy. They should have assistance in their household duties, and they should be instructed to go to bed at the first sign of a respiratory infection. They should be carefully examined for early signs of heart failure at least every two weeks during the first half of pregnancy, and once a week thereafter. The earliest sign of failure is usually *persistent* rales at the lung bases, which may appear before the patient herself is aware that anything has gone wrong. Once failure has set in, the patient should be put to bed and kept there under hospital conditions of absolute rest until she is delivered. If failure comes on during the first four months, it is extremely unlikely that the patient can be brought to term with a living baby, and it is well to advise interruption at this time. If failure does not occur until the sixth month or later, every effort should be made to tide the patient over with digitalization and absolute rest until she goes into labor, and then to let her deliver spontaneously from below. It is not advisable to give digitalis unless failure is present or appears to be impending. Surgical interference should never be attempted while the patient is in failure. The maternal mortality under such conditions is 50 per cent. If the uterus is not interfered with and severe congestive failure persists, the patient is apt to go into labor spontaneously, and it is the lesser risk to allow her to do so with the operator standing by to make the second stage as brief as possible as soon as the cervix is fully dilated.

So far as anesthesia is concerned, particular care should be taken to avoid anoxia and excitement. Ether and oxygen anesthesia in skilled hands is very satisfactory.

Under careful supervision the maternal mortality in heart disease can be reduced to nearly 2 per cent.

COMMITTEE ON STATE AND NATIONAL LEGISLATION

House Bill 1222, a bill proposing a separate board of registration for chiropractors will soon be brought before the House of Representatives.

This type of bill always has a good deal of active support, and your Committee on State and National Legislation strongly urges all fellows to write to their representatives and senators, asking them to work and vote against the bill.

The following bill is scheduled for hearing at the State House during the week of March 17:

March 18

H. 1618 (Public Health). Petition of D. N. Potter and others that certificates of vaccination or nonvaccination shall no longer be required as a prerequisite to attendance in the public schools or other institutions of the Commonwealth. The perennial antivaccination bill. *Opposed.*

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning March 16:

BERKSHIRE

Thursday, March 20, at 4:30 p.m., in the Bishop Memorial Building, Pittsfield. Dermatitis and Ec-

zema. Instructor: Maurice M. Tolman. Harry G. Mellen, *Chairman.*

BRISTOL SOUTH (Fall River Section)

Tuesday, March 18, at 4:30 p.m., at the Union Hospital, Fall River. Recent Advances in Medical Therapeutics. Instructor: Clifford L. Derick. Howard P. Sawyer, *Chairman.*

FRANKLIN

Thursday, March 20, at 8:00 p.m., in the Library of the Franklin County Public Hospital, Greenfield. Nutritional Deficiencies and the Uses of Preparations of Vitamins. Instructor: Harold J. Jeghers. Halbert G. Stetson, *Chairman.*

HAMPDEN

Wednesday, March 19, at 4:00 p.m., at the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:00 p.m., in the Outpatient Department of the Skinner Clinic, Holyoke Hospital, Holyoke. Chemotherapy in the Treatment of Gonococcal Infection. Instructor: Sylvester B. Kelley. Alfonso A. Palermo, *Chairman.*

HAMPSHIRE

Thursday, March 20, at 4:15 p.m., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Technic and Treatment of Primary, Secondary and Tertiary Syphilis. Instructor: William P. Boardman. Robert C. Byrne, *Chairman.*

WORCESTER

Tuesday, March 18, at 8:30 p.m., in the Nurses' Home of the Milford Hospital, Milford. Technic and Treatment of Primary, Secondary and Tertiary Syphilis. Instructor: C. Guy Lane. Joseph Ashkins, *Chairman.*

WORCESTER NORTH

Friday, March 21, at 4:30 p.m., in the Nurses' Home of the Burbank Hospital, Fitchburg. Obstetric Infections: Diagnosis and treatment. Instructor: Judson A. Smith. George P. Keaveny, *Chairman.*

DEATHS

FRANCIS — CARLETON S. FRANCIS, M.D., of Brookline, died March 8 at Kerrville, Texas. He was in his seventy-sixth year.

Born in Brookline, he attended Harvard University and received his degree from the Harvard Medical School in 1892. He studied abroad for a year and then opened an office in Brookline.

He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow and two sons survive him.

WETHERELL — BRYANT D. WETHERELL, M.D., of Boston, died March 5. He was in his fifty-third year.

Dr. Wetherell attended Williams College and received his degree from Harvard Medical School in 1918. He was a fellow of the Massachusetts Medical Society and the American Medical Association, and was on the staff of the Massachusetts General Hospital.

His widow survives him.

MISCELLANY

THE MEDICAL LIBRARY ASSOCIATION

Every physician who recognizes the important place that medical literature has in the rapid march of scientific progress will look with approval and gratification upon the splendid work of the Medical Library Association. Composed of all medical libraries of any consequence in the United States, and many in Canada and abroad, this group has long been committed to the development of medical libraries and to the better organization of library resources so that they may be of the greatest possible help in the furtherance of medical research and achievement. It goes without saying that the individual libraries and librarians that constitute the larger part of the membership have been of tremendous assistance to physicians and others in their daily investigations and experiments; hence, the association merits the support of physicians.

Organized some forty-three years ago, the Medical Library Association has become pre-eminent in its field and enjoys a well-earned international reputation. Dr. G. M. Gould, who was its founder, was also its first president. Succeeding him in this office were such illustrious men and women as Osler, Chadwick, Jacobi, Dock, Musser, C. Perry Fisher, J. C. Wilson, F. R. Packard, Lt. Col. C. C. McCulloch, H. L. Taylor, Browning, Garrison, Farlow, Barker, Wylde, Tice, Ruhrah, Malloch, Steiner, Miss M. C. Noyes, C. Frankenberger, W. W. Francis and J. F. Ballard. Colonel Harold W. Jones, of the Army Medical Library, is now president.

Perhaps the most outstanding activity of the association is its maintenance of an "exchange" — a clearing house through which members may exchange their duplicate books and journals with other libraries, so that they may find a place where they are needed. Every member library has profited by this mutually helpful arrangement, and frequently member institutions are able to complete series of great value.

Libraries having at least 1000 volumes of medical interest and open for regular use with a full-time librarian on duty are eligible for membership.

Officers and members of the association have worked untiringly toward the achievement of its numerous objectives, but it is admitted that support from dues alone is insufficient, owing to the limited number of medical libraries that are eligible for membership. As a consequence, the membership committee of the association is endeavoring to attract a larger number of "supporting members" from among its physician friends and others. A supporting member may be any person interested in medical libraries and medical literature. The dues are $5.00 annually; this includes the interesting quarterly *Bulletin of the Medical Library Association.* A supporting member has the privilege of attending the annual meetings and of making suggestions for the association's program. Dr. A. H. Sanford, of the Mayo Clinic, recently has contributed an article (The supporting member. *Bull. M. L. A.* 28:100–104, 1939), which points out some of the advantages accruing to the physician who participates in the work of the association through this type of membership.

The Medical Library Association is striving to create a stronger bond with the medical profession and to derive mutual benefit from a better understanding of its manifold problems. Further information and application blanks may be had from Miss Louise Williams, Chairman, Membership Committee, Old Capitol, Jackson, Mississippi.

REPORT OF MEETING

BOSTON ORTHOPEDIC CLUB

A regular meeting of the Boston Orthopedic Club was held at the Boston Medical Library on December 18, 1940. Dr. Fritz Teal, of Lincoln, Nebraska, spoke on the Orr method of treating compound fractures. The speaker recounted how Dr. Orr decided on his procedure. In studying war wounds in World War I, he concluded that immobilization of a fractured limb was best carried out by plaster of Paris and that the true goal of asepsis is the exclusion of infection rather than the discovery of effective bactericides. This original principle of Lister has often been lost sight of in many methods of caring for infection, such as the Carrel–Dakin treatment.

The Orr method consists in following the first fracture principles of good apposition and adequate immobilization. The fundamental aseptic principle of excluding further infection is carried out. And finally the technic of Paré — whereby the natural reparative properties of the body are allowed to carry out their work — is adopted. It is a simple, effective, timesaving method that may be employed in already infected as well as new cases. Dr. Teal stated that there is a decrease in the time of healing, better apposition, lack of introduction of bacteria by irrigation, failure of proteolysis and absence of evaporation and dehydration. It is not applicable to all cases, a noteworthy exception being those in which there is extensive soft-tissue damage.

In reviewing statistics of World War I, Dr. Teal remarked that 50 per cent of all fractures of the femur, tibia and humerus resulted in disability eight years later. He stated that although the use of Thomas splints and Dakin irrigations resulted in some improvement, the results still fell short of the optimum.

In contrast were cited the results in the recent Spanish War, where two different reports indicated a mortality of only about 3 to 5 per cent in cases of compound fracture. Furthermore, the number of amputations necessary was only 4 out of 1100 in one report and none in 5000 in the other. That gas gangrene is definitely decreased in the Orr treatment, even when bacteria may be demonstrated, was substantiated by the finding of this complication in only 1 of the 5000 cases thus treated.

Dr. Teal reviewed briefly the technic of the Orr treatment. The injured extremity is thoroughly cleaned with soap and water and then prepared with iodine around the lacerated area only. The patient is placed on a fracture table, although this is not necessary, and proper alignment is obtained by traction and assured by fluoroscopic examination. The field is then draped and widely débrided. Irrigation was found necessary only rarely. The opening is enlarged as necessary. Fixation pins are placed as soon as cleansing and débriding are complete. No sutures are employed. Vaseline gauze is then placed in the wound, although any similar type of dressing may be used. A sterile dressing, sheet wadding and plaster of Paris are then applied, and there is rarely any reason to remove these for inspection, for the clinical chart will furnish the necessary clues to infection. After four to six weeks a window is made in the cast under aseptic precautions. The cast may be changed if secondary infection is present. It has been found that the wound is usually healed by the time of fracture healing.

At the Orr clinic, good results are obtained in 84 per cent of cases, despite the fact that almost half the patients are referred to the clinic after previous treatment. In 6 cases, amputation, although advised elsewhere, was not performed, and 5 of these patients made a satisfactory recovery.

The discussion was opened by Dr. Henry Marble, of the Massachusetts General Hospital, who defended the use of the Thomas splint and the Carrel–Dakin treatment as carried out during World War I. The patients always arrived at the base hospitals in good condition, the wounds were secondarily sutured, and the dressings were neither painful nor time consuming. One objection to the Orr method was that plaster does not hold the fracture well, especially when it involves the tibia and femur, with the result that traction must often be resorted to. The incorporation of pins has corrected this, it was admitted. Other points not considered in the Orr treatment are the function of neighboring joints and the atrophy of muscle.

Dr. Otto Hermann, of the Boston City Hospital, emphasized that any method properly carried out usually gives good results. Sherman, of Pittsburgh, over a period of twenty-three years treated over 11,000 cases by débriding, irrigating with Carrel–Dakin solution and fixing by external or internal methods, as indicated. Of 630 fractures of long bones there were 23 amputations, no cases of sepsis and 2 deaths — from embolus and shock. Dr. Hermann concluded that the Orr method may prove efficacious in wartime, but that Sherman's methods seem better when time and space are available. At the Boston City Hospital, primary suture is carried out if the wound is less than eight hours old, and if a careful history shows that the place and manner of injury are not contraindications to such a procedure. A soap-and-water scrub is carried out for fifteen to twenty minutes. Irrigation is used only after the wound edge has been débrided. The type of fixation depends on the demands of the individual patient.

Dr. William Rogers, of the Massachusetts General Hospital, reiterated the importance of immobilization and surgical nonintervention. He believes, however, that vitallium plates, especially when combined with traction, afford better immobilization than plaster, even when pins are incorporated; this also allows uninterrupted observation. The wound is left alone until walled off by granulation tissue, whether promoted by vaseline gauze or some other means. Bone chips should be left in place. Chemotherapy is employed whenever indicated.

Dr. Carl Walter, of the Peter Bent Brigham Hospital, stated that no one routine was carried out at that institution, the usual method being to cleanse thoroughly, carry out primary suture when possible and treat the wound as an ordinary fracture. The few deaths were from indirect causes, and amputations only for gas gangrene. Dr. Walter emphasized that Lister was primarily interested in asepsis as opposed to antisepsis, one of his first reported cases being a compound fracture that received treatment essentially similar to the Orr method. Lister stressed the importance of nonintervention and the wide spacing of any dressings, and then the changing of only the exterior ones until healing has taken place.

The discussion was continued by Dr. Champ Lyons, of the Massachusetts General Hospital, who presented his views on chemotherapy, especially as related to anerobic infection. Anerobic bacteria are relatively frequent contaminants, and the duration or mode of accident does not ensure against their presence. Statistics are as yet inconclusive as to the effect of the newer chemicals on the gas-forming organisms or their relative therapeutic effects. Sulfathiazole, it has been demonstrated at Johns

Hopkins Hospital, deters sound wound healing, and inhibits the growth of epithelium and fibroblasts in a concentration of 20 mg. per 100 cc. Sulfanilamide, on the other hand, has occasioned no trouble with healing in concentrations as high as 200–400 mg. per 100 cc. Key has used sulfanilamide powder in wounds covered only by skin. Eight grams will result in a blood-stream level of 12 to 14 mg., which rapidly declines unless auxiliary oral therapy is started in twelve hours. Sulfapyridine experimentally is more effective against established gas-bacillus infection than sulfanilamide, especially when serum is being used. Since it requires too long to determine which bacterium is at fault, the use of trivalent antiserum was advised. The initial therapeutic dose should be 120,000 units of *Clostridium welchii* and *Cl. oedematis maligni* antitoxins and 30,000 units of that for the other gas-forming bacilli. This is the amount contained in four therapeutic vials, and should be followed by two vials every eight hours until improvement occurs. A sulfapyridine level of 8 mg. per 100 cc. should be maintained.

In closing the discussion, Dr. Teal explained that Dr. Orr had been using pins for fifteen years, and that this was considered simpler than, and as effective as, plating. The latter was conceded to be satisfactory if left open. Primary suturing was found to invite too much trouble, especially since the wounds heal as soon as the fracture and can be improved by secondary plastic operations, if necessary.

NOTICES

MEDICAL AND SURGICAL SUPPLY COMMITTEE

It will help England greatly in the care of victims of bombing raids and help England prepare for the coming invasion, if physicians in this country will give discarded or superfluous surgical instruments or surgical dressings.

Any such material should be sent to the Medical and Surgical Supply Committee, 420 Lexington Avenue, New York City.

COMMUNITY NURSING COUNCIL OF BOSTON

The third annual meeting and luncheon of the Community Nursing Council of Boston will be held at Perkins Hall, Women's Educational and Industrial Union, 264 Boylston Street, Boston, on Thursday, March 27, at 12:30 p.m.

PROGRAM

Business meeting.
Nursing in National Defense. Miss Stella Goostray, R.N.
The Federal Census of Registered Nurses. Miss Helene G. Lee, R.N.
Progress of Legislation. Miss Margaret Dieter, R.N.

The charge for luncheon will be 75 cents. Reservations, sent to the Community Nursing Council of Boston, 80 Federal Street, Boston, should be made by Monday, March 24.

CARNEY HOSPITAL

The monthly clinical meeting and luncheon of the Carney Hospital will be held in the auditorium of the Carney Hospital on Monday, March 24, at 11:30 a.m.

PROGRAM

Nasal Allergy. Dr. William T. Haley.
Review of Cases Requiring Hysterectomy at Term. Dr. William J. McDonald.
Acute Cor Pulmonale. Dr. Sylvester McGinn.
Surgical Diseases of the Breast. Dr. John W. Spellman.

Physicians and medical students are invited to attend.

BOSTON LYING-IN HOSPITAL

There will be a meeting of the Journal Club of the Boston Lying-in Hospital on Wednesday, March 19, at 8:15 p.m. Dr. Edward G. Waters, assistant clinical professor of obstetrics and gynecology, Columbia University College of Physicians and Surgeons, will speak on "Local and Spinal Anesthesia in Obstetrics."

Physicians and medical students are cordially invited to attend.

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

There will be a meeting of the New England Society of Physical Medicine at the Hotel Kenmore, Boston, on Wednesday, March 19, at 8:00 p.m. A council meeting at 6:00 p.m. will be followed by an informal dinner in the Empire Room at 6:30 p.m.

PROGRAM

Symposium on the Treatment of Arthritis.
The Parenteral Use of Amiodoxyl Benzoate. Dr. Albert G. Young.
Mecholyl Iontophoresis. Dr. Wilmot L. Marden.
Diathermy. Dr. William D. McFee.
X-ray. Dr. Herman A. Osgood.
Discussion by Dr. A. Carleton Potter.

All members of the medical profession are cordially invited to attend.

NEW ENGLAND HEART ASSOCIATION

There will be a meeting of the New England Heart Association on Monday, March 24, in the pathological amphitheater of the Boston City Hospital at 8:15 p.m.

PROGRAM

Familial Occurrence of Congenital Cardiac Anomalies. Drs. Gerald C. Walker and Laurence B. Ellis.
The Influence of the Climacteric on the Course of Organic Heart Disease. Drs. Burton E. Hamilton and Paul Kunkel.
Paroxysmal Ventricular Tachycardia: A study of 33 cases. Dr. Conger Williams.
The Significance of the Wandering Auricular Pacemaker. Dr. Andrew M. Burgess, Jr.
The Occurrence of Sinus Arrhythmia in Association with Acute Myocardial Infarction. Dr. Carey M. Peters.
Nonarterial Disorders Simulating Diseases of the Peripheral Arteries. Dr. Edward A. Edwards.

Interested physicians and medical students are cordially invited to attend.

NEW ENGLAND PATHOLOGICAL SOCIETY

The next meeting of the New England Pathological Society will be held at the Children's Hospital on Thursday, March 20, at 8 p.m.

PROGRAM

Survey of the Bacteriological Laboratories in Massachusetts. Dr. Leo Rane.

Co-operation between the Pathologist and the Embalmer. Mr. Austin W. Martin.

What Deaths Are Medico-Legal? Dr. William J. Brickley.

Business meeting.

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY

The regular spring meeting of the New England Oto-Laryngological Society will be held on Wednesday, March 19, at the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, at 4 p.m. The evening program will be devoted to a symposium on "The Management of Some of the Complications Arising from Acute and Chronic Otitis Media." Discussion will follow by Drs. Harold Tobey, Charles T. Porter, Maxwell Finland, Champ Lyons, W. Jason Mixter and Charles S. Kubik.

NORFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the Norfolk District Medical Society will be held at the Hotel Puritan, Boston, on Tuesday, March 25, at 8 p. m.

PROGRAM

Business.

Nutritional Deficiencies and the Uses of Preparations of Vitamins. Dr. Harold J. Jeghers.

Discussion.

Collation.

26TH DIVISION MEDICAL SERVICE

The 26th Division (Massachusetts) is rapidly filling to war strength with men from almost every walk of life. One outstanding numerical exception has been the representation of the Massachusetts Medical Society.

More qualified physicians are required to fill vacancies in the Division Medical Service. The requirements are: a medical diploma from a Class-A School, a minimum of one year's internship and a sound physique. The candidate must also be no more than thirty-five years of age. Previous military training is desirable but not essential.

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS AND AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

The annual meeting of the American Association of Industrial Physicians and Surgeons and the American Industrial Hygiene Association will take place from May 5 to 9 at the Hotel William Penn, Pittsburgh, Pennsylvania. Industrial health specialists from all parts of North and South America will attend. An intensive graduate course in present-day industrial health problems will be presented. May 5 will be devoted to a series of dry clinics in industrial medicine and surgery at the Mercy Hospital. For detailed information write to Mr. A. G. Park, executive secretary, 540 North Michigan Avenue, Chicago, Illinois.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING SUNDAY, MARCH 16

MONDAY, MARCH 17

12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

TUESDAY, MARCH 18

*9–10 a.m. Syphilis and the Spinal Fluid. Dr. W. A. Hinton. Joseph H. Pratt Diagnostic Hospital.

*12 m. Asthma: Its treatment. Dr. Francis M. Rackemann. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

12:15–1:15 p.m. Clinicoroentgenologic conference. Peter Bent Brigham Hospital amphitheater.

WEDNESDAY, MARCH 19

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*12 m. Clinicopathological conference. Children's Hospital.

4 p.m. New England Oto-Laryngological Society. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston.

*8 p.m. New England Society of Physical Medicine. Hotel Kenmore, Boston.

*8:15 p.m. Boston Lying-in Hospital. Journal Club meeting.

THURSDAY, MARCH 20

*8:30 a.m. Combined clinic of the medical, surgical, orthopedic and pediatric services of the Children's Hospital and the Peter Bent Brigham Hospital, at the Children's Hospital.

*9–10 a.m. Tobacco and Peripheral Vascular Disease. Dr. J. S. Sprague. Joseph H. Pratt Diagnostic Hospital.

11 a.m.–3:30 p.m. Massachusetts Tuberculosis League. University Club, Trinity Place and Stuart Street, Boston.

8 p.m. New England Pathological Society. Children's Hospital, Boston.

FRIDAY, MARCH 21

*9–10 a.m. Arthritis clinic. Dr. W. Bauer. Joseph H. Pratt Diagnostic Hospital.

SATURDAY, MARCH 22

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*Open to the medical profession.

MARCH 21–22 — New York University College of Medicine, Alumni Day. Page 135, issue of January 16.

MARCH 23 — Free public lecture, Quincy City Hospital. Page 436, issue of March 6.

MARCH 24 — New England Heart Association. Page 483.

MARCH 24 — Carney Hospital. Page 483.

MARCH 26 — Tufts College Medical School Alumni Association. Page 348, issue of February 20.

MARCH 27 — Community Nursing Council of Boston. Page 483.

MARCH 31–APRIL 4 — Sixth Annual Postgraduate Institute of the Philadelphia County Medical Society. Page 349, issue of February 20.

APRIL 2–4 — New England Health Institute. Page 398, issue of February 27.

APRIL 10 — Pentucket Association of Physicians. Page 263, issue of August 15.

APRIL 21–25 — American College of Physicians. Page 1065, issue of June 20.

APRIL 28–30 — American Academy of Physical Medicine. Scientific session. Hotel Pennsylvania, New York City.

MAY 5–9 — American Association of Industrial Physicians and Surgeons and American Industrial Hygiene Association. Notice above.

MAY 21, 22 — Massachusetts Medical Society, Boston.

MAY 28–JUNE 2 — American Board of Obstetrics and Gynecology. Page 262, issue of February 6.

JUNE 2–6 — American Medical Association. Cleveland, Ohio.

OCTOBER 14–17 — American Public Health Association. Page 135, issue of January 16.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH

APRIL 2 — Pediatric Problems in General Practice. Dr. Joseph Garland. Addison Gilbert Hospital, Gloucester.

MAY 14 — Relation of the Doctor to the Law. Mr. Leland Powers. New Ocean House, Swampscott.

FRANKLIN

MAY 13 — This meeting will be held at 11 a.m. at the Franklin County Hospital, Greenfield.

NORFOLK

MARCH 25 — Page 484.

MAY 8 — Censors' meeting. Hotel Puritan.

SUFFOLK

APRIL 30 — Page 604, issue of October 10.

MAY 1 — Censors' meeting. Page 261, issue of February 6.

WORCESTER

APRIL 9 — Hahnemann Hospital, Worcester.

Supper at 6:30 p.m. followed by a business meeting and scientific program.

BOOK REVIEWS

Rose and Carless Manual of Surgery. Sixteenth American edition. Edited by William T. Coughlin, M.D. From the sixteenth English edition by Cecil P. G. Wakeley, D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.R.A.C.S.; and John B. Hunter, M.C., M.Chir. (Cantab.), F.R.C.S. 8°, cloth, 1656 pp., with 1034 illustrations and 30 colored plates. Baltimore: Williams and Wilkins Company, 1940. $9.00.

According to the author's preface, this ponderous text — it weighs 6½ pounds — has been thoroughly revised and the first eight chapters have been entirely rewritten. These chapters discuss general and specific infections, inflammation and repair, ulceration, gangrene, wounds, tumors, hemorrhage and shock. It seems reasonable to examine them to determine how much new material has found its way into the book.

Seven pages are devoted to the subject of shock, and the following statements appear in the text: "All observers are agreed that the constant feature [of shock] is the depression of the blood pressure from the normal systolic pressure of 110 to 120 mm. of hemoglobin to 50 or below. . . . An estimation of the color index of the blood may help [in diagnosis], since it is normal in pure shock, but is lowered following hemorrhage." The only reference to transfusion in the treatment of shock is as follows: "The actual transfusion of blood is often useful, but may be disappointing in its effect. It is most valuable in cases of shock associated with severe hemorrhage where the patient's condition is aggravated by anoxemia from the lowered hemoglobin. In pure shock the loss of blood volume is a loss of the fluid portion of the blood, and transfusion in these cases is of less value than the use of intravenous saline. The hemoglobin content of the blood may be taken as a guide; in shock a fall of hemoglobin below 70 per cent would form an indication for a blood transfusion."

In regard to the treatment of wounds, it is stated, "Tincture of iodine, either full strength or diluted half with 95 per cent alcohol, is in very general use for the first antiseptic treatment of wounds. The excess is removed from the wound by pouring in alcohol."

In the care of septic joints, following drainage, "the essential element is to urge, or if need be coerce, the patient into moving the joint actively, however painful it may be."

The progressive undermining ulcers due to microaerophilic streptococcal infections are described as "gangrene of the skin following operation," but there is no recognition of the extensive contributions that have been made in recent years to the etiology or therapy of these conditions. Although innumerable pharmaceutical preparations are recommended in treating various conditions, zinc peroxide finds no place in the index, and is referred to only once in the text in connection with the treatment of actinomycosis. Sulfanilamide is given nine references in the index; six of them to page numbers not present in the book. The remaining three briefly discuss the use of the drug in streptococcal and gonococcal infections, with a footnote reference to articles by the staff of the Wellcome Research Laboratories in the *Lancet* in 1936 and 1937.

Postoperative parotitis "today is very rare, since oral sepsis is always treated before any abdominal operation is contemplated. Treatment should be prompt, incision and drainage being required."

These quotations have been selected deliberately to emphasize the wide divergence that apparently exists between American and English teaching in surgery, and to point out how difficult it is to find a text that can be carried through numerous editions and still be up to date, with elimination of antiquated teachings and inclusion of new material. Similar quotations and omissions could be culled from most of the other chapters. Differences in terminology further complicate the picture and confuse the student. Thus, for example, benign giant-cell tumor of bone is described as "myeloma" or "osteoclastoma." In the treatment of osteogenic sarcoma, the authors state that "x-ray treatment alone often causes a disappearance of the tumor, but recurrence is the rule."

Nearly 400 pages are devoted to orthopedic problems and diseases of bones, joints, and tendons. Superficial inspection of this section reveals an illustration of Colles' fracture in a cast in the cock-up position, and a bone plate applied in reduction of a fractured femur, held in place with screws which extend only to the medullary cavity. The use of a pylon following amputation is condemned; there is apparently no appreciation of the mechanism of Volkmann's contracture, and passive movements of joints are strongly recommended in the restoration of function following injuries.

It is perhaps unnecessary to bring up more examples of omissions, inaccuracies, and errors in the book. Some of the sections are sound. There are numerous illustrations, and for the most part they are helpful in illuminating the text. In summary, however, it is the reviewer's opinion that a student would be ill advised to acquire or to depend on this text.

Juvenile Delinquents Grown Up. By Sheldon and Eleanor Glueck. 8°, cloth, 330 pp., with appendixes A, B and C and 85 tables. New York: The Commonwealth Fund, 1940. $2.50.

This is unquestionably one of the most important objective and scientific discussions of juvenile delinquency to appear in recent years, and gives the Gluecks first rank among the criminologists of the world. The reader may recall that in 1934 they published *One Thousand Juvenile Delinquents*, a follow-up study to determine the effectiveness of treatment.

In this book the authors make a painstaking and searching study of the same delinquents for the second and third five-year periods, directing their attention to such questions as, What has happened since? Have the delinquents gone from bad to worse, or has the picture brightened? How can one account for the changes in behavior? How far can delinquency be predicted? The thoroughness and ingenuity employed in ferreting out many of these elusive characters make interesting reading, and document one angle of the Gluecks' scientific thoroughness.

The book consists of twenty-two chapters; it discusses the characteristics of delinquents, follows them through the three periods and compares reformed and unreformed, the young and the old and the serious and the minor offenders. The book makes a strong case for the possibility

of scientific prediction if judges, parole officers and boards would only begin the experimental use of the tables furnished.

With the passage of years there has been a steady diminution in the number of youths who continue to be offenders. By the age of twenty-nine almost 40 per cent have ceased to be criminals. Even among those who continued their criminal careers, significant improvement was noted. However, the chief cause of the improvement was not the penologic treatment but a natural process of maturation. For, regardless of the age of the offenders at the time delinquency began, it ran a fairly steady and predictable course. There is a marked resemblance in delinquent or criminal behavior at a point equidistant from the onset of misconduct, regardless of the age of the persons concerned.

Comparison of delinquents who reformed within fifteen years with those who had not reformed showed that the former had better heredity and more wholesome environment than the men who continued criminal careers. Those who reformed before twenty-one possessed both better innate equipment and environmental advantages. The more serious offenders had a worse congenital equipment and were reared in even less favorable circumstances than those responsible for minor offenses.

Four conduct types are distinguished, and prediction tables are offered that should make sentencing more intelligent if judges would experimentally sentence every other offender on the basis of it and study the results.

The authors conclude, "It is not so much arrival at any particular age-span . . . as the achievement of a degree of maturity, that makes for social adaptation on the part of former delinquents." Delinquents are primarily infantile; they mature much more slowly. Too often they reform by wearing out. As they get older they resort to minor crimes. They lose courage and do not have the physical equipment for more daring escapades.

This book will repay study by physicians, psychiatrists, sociologists and counselors of youth. It should be "must" reading for criminologists, judges and members of parole boards.

The Treatment of Diabetes Mellitus. By Elliott P. Joslin, M.D., Sc.D.; Howard F. Root, M.D.; Priscilla White, M.D.; and Alexander Marble, M.D. Seventh edition, thoroughly revised. 8°, cloth, 783 pp., with 117 tables. Philadelphia: Lea and Febiger, 1940. $7.50.

This familiar and valuable treatise now appears in its seventh edition under the coauthorship of Drs. Joslin, Root, White and Marble. The revision of this study of 18,000 of their own cases, as well as bringing the ever-enlarging literature in this field up to date, is a task of no mean proportions, but it has been done thoroughly and effectively since the appearance of the sixth edition in 1937.

The increased frequency of allergic phenomena since the introduction of protamine insulin has led to a new chapter on allergy in diabetes by McDaniel, and the increased interest in hyperinsulinism has led to a chapter on this subject by Marble. A most valuable chapter is that on the physiology of diabetes, revised by Marble, which includes references to all the newer developments in this field, including the important work of Houssay, Young, Himsworth and many others.

There has been almost no important change in the treatment of diabetes in the last three years, but the study of the cases that have been treated with protamine insulin for three years has led to a more standardized use of this important therapeutic agent. A diagrammatic outline of standard treatment such as appears on page 288 is the result of the observation of many thousand cases. Little has been changed in diet therapy, but the newer knowledge of vitamins has not been neglected and, in fact, appears in this volume in a summarized form that is most convenient, entirely apart from its value in treating diabetic patients.

Coma is still considered to be present whenever the carbon dioxide content is below 20 vol. per cent, whether or not the patient shows any disturbance of consciousness, although this method of classification has been the object of criticism from several sources. Whatever criteria are used, however, the mortality rate for acidosis cases treated by these authors remains a challenge for others to equal.

Dr. White's work with pregnant diabetic patients, particularly the prolan studies and estrin and progestin therapy carried on in conjunction with Smith and Smith, is most interesting, and the results of this treatment are most encouraging. Whether these results have been confirmed in other clinics is not stated.

The volume remains the standard work on diabetes mellitus.

Rheumatic Fever: Studies of the epidemiology, manifestations, diagnosis and treatment of the disease during the first three decades. By May G. Wilson, M.D. 4°, cloth, 595 pp., with 47 illustrations, 76 figures, 81 tables and 31 charts. New York: The Commonwealth Fund, 1940. $4.50.

Of the economic importance of rheumatic fever in this part of the world there is no question whatsoever. An increasing amount of study has lately been devoted to it—Dr. Wilson herself has been interested in the subject for more than two decades. Although a great deal has been learned, there still remains more that has not. The most elaborate treatise of today, therefore, can represent only a sort of stocktaking of knowledge. Such stocktaking, however, is extremely valuable.

The work begins with a brief historical summary; then follow chapters on epidemiology, including familial epidemiology, on etiologic hypotheses and on bacteriologic studies. Part II embraces general signs and symptoms, heart involvement and involvement of other organs and tissues, and bacterial endocarditis. Part III covers the age and type of manifestation at onset, prognosis and life expectancy. Part IV discusses the criteria of diagnosis and differential diagnosis, including the regression of physical signs, radioscopic examination of the heart, and the value of exercise tests, of the measurement of vital capacity and of electrocardiography. Part V is devoted to the care, management and therapy of the rheumatic child. The discussions throughout are not merely general; they are illuminated and reinforced by appropriate case histories and are illustrated with charts, plates and tables based on actual experience. Moreover there are extensive bibliographies.

It is not necessary that every doctor should own this book, not even every pediatrician or cardiologist. But it should be in every medical library, and in the libraries of all hospitals where research is undertaken. For such a book of reference often saves repetitious work, or better still, may serve as the steppingstone to new and even more definitive studies.